NEIKKURI EXAMINATION IN ATHI NURAI NEER / FROTHY URINE A CONDITION OF ALBUMINURIA

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By
Dr. GAYATRI. R
PG Scholar
National Institute of Siddha
Chennai-47

Under the guideship of
Dr. G.J.Christian M.D (S)
HOD (i/c)
National Institute of Siddha
Chennai-47

Study Centre

Dept. of Noi Naadal,
National Institute of Siddha
Tambaram Sanatorium, Chennai – 47.

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DECLARATION BY THE CANDIDATE

I hereby declare that this Dissertation entitled ***"NEIKKURI EXAMINATION IN ATHI NURAI NEER / FROTHY URINE A CONDITION OF ALBUMINURIA"*** is a bonafide and genuine research work carried out by me under the guidance of **Dr. G.J.Christian M.D (S)** HOD (i/c), Dept of Noi Naadal, National Institute of Siddha, Chennai – 47, and the dissertation has not formed the basis for the award of any other degree, Diploma, Fellowship or other similar title.

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Date: 29.06.2018 Signature of the Candidate

BONAFIDE CERTIFICATE

Certified that I have gone through the dissertation submitted by **Dr. Gayatri.R (Reg.No: 321515201)** a bonafide student of final year M.D(s), Branch-V, Department of Noinaadal, National Institute of Siddha, Tambaram Sanatorium, Chennai - 47, and the dissertation work has been carried out by the individual only. This dissertation does not represent or reproduce the dissertation submitted and approved earlier.

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INDEX

1 : INTRODUCTION

Siddha medicine considers human body as the direct replica of nature. With much praise to the divine, quote *"Andathil ullathey pindam, pindathil ullathey andam"* stated by *Siddhar Sattaimuni* there are so many *Siddhar* school of thoughts in high class T*amizh* which quantifies the theories of human body origin expressed in material anatomy called *Panchabootha Pancheekaranam*, its evolution , Physiology, Transformation and finally degeneration. All this phenomenon comes under the eternal basic principles called 96 *Thathuvam* or constituent principles including Physical, psychological and spiritual entities of a human-being. The *Siddhars* School fully recognizes these ninety-six *thathuvam* and further added that human body is composed of 72,000 *Naadi, Dasa Naadi, Dasa Vaayus* all together in the network; and it is owing to the derangement of the any of the principles one become liable to diseases.

Siddha system of medicine is having a well-established protocol for diagnosing diseases. It is mainly based on 96 *thathuvam* among them *Tri-humoural* theory is mainly used for classifying, diagnosing a disease and treating a patient. When compared to other diagnostic techniques prevalent in *Siddha, Neerkkuri* and *Neikkuri* seem to be concordant.

Urine is actually a fluid 'biopsy' marker of kidney ailments and provides a fountain of information. Kidney is the only organ with such a convenience of non-invasive means to evaluate its status. In addition, because urine is an ultra- filtrate of plasma, it can be used to evaluate and monitor body homeostasis and many metabolic disease processes. *Siddhars* had developed urinalysis without the help of any sophisticated instruments on those days and they named it as *Siruneer sodhanai* (*Neerkkuri and Neikkuri).* Ancient *Siddhars* have not outlined analytical methods and parameters of research that a modern scientist might understand; however, they have laid down procedures and methods in there on way. *Siddhars* generally examined urine samples based on *Niram (*colour), *manam (*odour*), edai* (Specific gravity), *nurai (*Froth), *enjal* (Deposits) and classified urine samples according to colour variation, tri-humoural variations as *Vatham, Pitham* and *Kabam neer etc.* After assessment, *Neikkuri* (Oil on urine sign) will be conducted for diagnosis as well as prognosis. For *Vatha* urine, *Neikkuri* they have mentioned the spreading of oil drop like a serpent, for *Azhal neer* like a ring, and for *kabam neer* like a pearl . Therefore, there can be some unique features for *vatham, pitham, kabam* urine samples to bring their corresponding patterns.

Generally, *Siddha* system of medicine, which is having its own traditional and experienced time tested diagnostic techniques, but to create a wider acceptance of this system a scientific validation is required.

In *Neikkuri* Analysis the instilled oil drop is spreading in different patterns or standing as such, spreading of oil is due to gravity for equifying the weight of oil micelles and surface tension of urine makes the oil stay over urine(since its density is less than that of urine). Urine contains 94% water and 6% of solutes, water as a matter contains empty space, which solutes can occupy. Naturally, the spreading of oil will show different patterns with water the space of which is occupied by different solutes. So there may be specific solutes in *Vatham, Pitham* and *Kabam neer* which makes the the oil drop to spread in different patterns and the three types of urine will have three different surface tension and specific gravity to make the oil drop to spread or stay still.

Athinurai neer(Albuminuria*)* is a condition in which *avalambakam, kilethakam, bothakam, tharpagam*, and *santhigam* gets deranged which makes *vatham* excrete through urine which can be seen as froth. The importance of albuminuria as an independent predictor of progressive renal disease and cardiovascular mortality was realized in a number of prospective and epidemiological studies particularly in patients with diabetes and hypertension. In adults, the link between albuminuria, cardiovascular disease, and progressive renal disease is now well established in patients with systemic diseases including diabetes mellitus. Interestingly, microalbuminuria has also emerged to be an important risk factor for the development of cardiovascular disease, and all cause mortality in the general population. Faced with the realization of increasing prevalence of obesity, type 2 diabetes, and metabolic syndrome in children, screening for microalbuminuria seems highly relevant in the pediatric population to detect and prevent cardiovascular disease. Chronic Kidney Disease (CKD) conditions are also determined by the presence of reduced estimated Glomerular Filtration Rate (eGFR) or markers of kidney damage, generally determined by elevated albuminuria. Recent data have demonstrated that albuminuria is a strong and independent predictor of risk for mortality, cardiovascular disease, end-stage renal disease, acute kidney injury, and CKD progression.

This study aims in diagnosing derangement of *Vatham, Pitham, Kabam* humours and to find out any specific factors that leads to the change in the urine context and formation of specific patterns on instillation of gingelly oil over urine sample of patient with *Athi Nurai Neer (Albuminuria)*.And to develop a standard protocol for diagnosing albuminuria condition especially by neerkkuri and neikkuri technique, because the available golden standards to determine albuminuria are not cost effective.The findings may help for further scope in the field of Research and diagnosis through *Siddha* approach of Urine examination. So it will be useful if a standard tool to determine albuminuria by siddha diagnostic techniques with much accuracy is developed.

2.1. Primary objective:

- To document the diagnostic patterns of *Neikkuri* in *ATHI NURAI NEER* condition.

2.2. Secondary objective:

To observe for any significant *Neikkuri* pattern which may provide a clue in the diagnosis, prognosis or its complications.

3A SUGARANA NILAI IN SIDDHA MEDICINE (PHYSIOLOGY

The five basic elements, namely Aagayam (Space), Kaal (Air), Thee (Fire), Neer (Water), and Mann (Earth) are the building blocks of all the physical and subtle bodies existing in this whole universe. These are called as the 'Adippadai boothams' (Basic Elements) (or) 'Panchaboothams'.

These five elements altogether constitute the human body and also the origin of other materialised objects, explained as Pancheekaranam (Mutual Intra Inclusion). None of these elements could act independently by themselves. They could act only in co-ordination with other four elements. All the living creatures and the non-living things are made up of these five basic elements.

உலகம் ஐந்து பூதம்

"நிலம் நீர் தீ வளி விசும்போடைந்தும்
கலந்தமைக் கருவகம் மாதலின்"

- தொல்காப்பியம்

தேகம் ஐந்து பூதம்

"தலங்காட்டி இத்தச் சடமானஞ்சுதம்
நிலங்காட்டி நீர் காட்டி நிறஞ்சுந் தீ காட்டி
வலங்காட்டி வாயுவால் வளர்ந்தே இந்த
குலங்காட்டி வானில் குயாய் இருந்ததே"

- பதினெண்சித்தர் நாடி சாஸ்திரம்

As per the above lines, the universe and the human body are made of five basic elements.

A.THE 96 BASIC PRINCIPLES (96 THATHUVAM):

According to *Siddha* system of medicine, '*Thathuvam'* is considered as a science that deals with basic functions of the human body. Siddhars described 96 principles as the basic constituents of human body that include physical, physiological, psychological and intellectual components of an individual. These 96 Thathuvams are considersd to be the cause and effect of our physical and mental well-being. The Thathuvam is the author of the conception of human embryo on which the theory of medicine is based.

1. BOOTHAM – 5 (ELEMENTS):

- Mann - Earth
- Neer - Water
- Thee - Fire
- Vaayu - Air
- Aagayam - Space

2. PORI -5 (SENSORY ORGANS):

- Mookku (Nose) - It is a component of Mann bootham
- Naakku (Tongue) - It is a component of Neer bootham
- Kan (Eye) - It is a component of Thee bootham
- Thol (Skin) - It is a component of Vaayu bootham
- Kadhu (Ear) - It is a component of Aagayam bootham

3. PULAN -5 (FUNCTIONS OF SENSORY ORGANS):

- Nugarthal - Smell : It is a component of Mann bootham
- Suvaithal - Taste : It is a component of Neer bootham
- Paarthal - Vision : It is a component of Thee bootham
- Thoduthal - Touch : It is a component of Vaayu bootham
- Kettal - Hearing: It is a component of Aagayam bootham

4. KANMENTHIRIYAM – 5 (MOTOR ORGANS) AND KANMAVIDAYAM

- Vaai(Mouth) - Vasanam - Vaaku - The speech occur in relation with Space element
- Kaal (Leg) - kamanam- Paadham -The walking take place in relation with Airelement.
- Kai (Hands) - Dhaanam – Paani - Giving and taking are carried out with Fire element
- Eruvai (Rectum) - Visarkam- Paayuru -The excreta is removed in association with Water element
- Karuvai (Genital organ) - Aanandham – Ubastham - Sexual acts are carried out in association with Earth element.

5. KARANAM – 4 (INTELLECTUAL FACULTIES)

- Manam – Thinking about a thing
- Bhuddhi – Deep thinking or analyzing of the thought
- Siddham – Determination to achieve it
- Agankaaram – Achievement faculty

6. ARIVU – 1 (WISDOM OF SELF REALIZATION)

To analyse good and bad.

7. NAADI -10 (Channels of Life Force responsible for the Dynamics of Life energy**)**

- Idakalai – Starts from the right big toe and ends at the left nostril.
- Pinkalai – Starts from the left big toe and ends at the right nostril.
- Suzhumunai – Starts from moolaathaaram & extend upto centre of head.
- Siguvai – Located at the root of tongue, helps in swallowing food.
- Purudan – Located in right eye.
- Kanthari – Located in left eye.
- Aththi – Located in right ear.
- Alambudai – Located in left ear.
- Sangini – Located in genital organs.
- Gugu – Located in anorectal region.

8. VAAYU – 10 (Vital nerve force which is responsible for all kinds of movements**)**

- **PRANAN (UYIR KAAL):**

  This is responsible for the respiration of the tissues, controlling knowledge, mind and five sense organs and digestion of the food taken in.

- **ABANAN (KEEL NOKKU KAAL):**

  It lies below the umbilicus. It is responsible for the downward expulsion of stools and urine, ejaculation of semen and menstruation, child birth.

- **VIYANAN (PARAVU KAAL):**

  This is responsible for the motor and sensory functions of the entire body and the distribution of nutrients to various tissues.

- **UTHANAN (MEL NOKKU KAAL):**

  It originates at utharakini. It is responsible for digestion, absorption and distribution of food. It is responsible for all the upward movements.

- **SAMANAN (NADUKKAL):**

  This is responsible for the neutralization of the other 4 valis, i.e. Pranan, Abanan, Viyanan and Uthanan. Moreover it is responsible for the nutrients and water balance of the body.

- **NAAGAN:**

  It is a driving force of eye balls and responsible for their movements.

- **KOORMAN:**

  It is responsible for the opening and closing of the eyelids and also vision. It is responsible for yawning.

- **KIRUKARAN:**

  It is responsible for the salivation of the tongue and also nasal secretion. Responsible for cough and sneezing and induces hunger.

- **DEVATHATHAN:**

  This aggravates the emotional disturbances like anger, lust and frustration etc. As emotional disturbance influence to a great extent the physiological activities, it is responsible for the emotional upsets.

- **DHANANCHEYAN:**

  Expelled after 3 days of death by bursting out of the cranium. It is responsible for edema, plethora and abnormal swellings in the body in the pathological state.

9. ASAYAM – 5 (VISCERAL CAVITIES):

- **Amarvasayam** (Reservoir organ): Stomach (digestive organ). It lodges the ingested food.
- **Pakirvasayam** (Digestive site): Small intestine. The digestion of food, separation and absorption of saaram from the digested food are done by this asayam.

- **Malavasayam** (Excretory organ for the solid waste): Large Intestine, especially rectum. Responsible for the expulsion of undigested food parts and flatus.
- **Salavasayam** (Excretory organ for the liquid waste): Urinary bladder, kidney. Responsible for the formation and excretion of urine.
- **Sukkilavasayam** (Genital organs): Place for the formation and growth of the sperm and ovum.

10. KOSAM – 5 (FIVE STATES OF THE HUMAN BODY OR SHEALTH):

- Annamaya Kosam – Physical Sheath (Gastro intestinal system)
- Pranamaya Kosam – Respiratory Sheath (Respiratory system)
- Manomaya Kosam – Mental Sheath (Cardio vascular system)
- Vignanamaya Kosam – Intellectual Sheath (Nervous system)
- Anandhamaya Kosam – Blissful Sheath (Reproductive system)

11. AATHARAM – 6 (STATIONS OF SOUL):

- **MOOLADHARAM :**

  Situated at the base of the spinal column between genital organ and anal orifice. Letter "ஓம்" is inscribed.

- **SWATHITANAM :**

  Located 2 finger breadths above the Mooladharam, (i.e) between genital and naval region. Letter "ரு" is inscribed. Earth element attributed to this region.

- **MANIPOORAGAM :**

  Located 8 finger breadths above the Swathitanam, (i.e) at the naval center. Letter "மி" is inscribed. Element is Water.

- **ANAKATHAM :**

  Located 10 finger breadths above Manipooragam, (i.e) location of heart. Letter "சி " is inscribed. Element is Fire

- **VISUTHI :**

  Located 10 finger breadths above the Anakatham (i.e) located in throat. Letter "வா" is inscribed. Element is Air.

- **AAKINAI :**

  Located between two eyebrows. Element is Space. Letter "யீ" is inscribed.

12. MANDALAM- 3 (REGIONS):

- **Thee Mandalam** (Agni Mandalam) Fire zone
  Fire Region, found 2 fingers width above the Mooladharam.
- **Gnayiru Mandalam** (Soorya Mandalam) Solar zone
  Solar Region, located with 4 fingers width above the umbilicus.
- **Thingal Mandalam**(Chandra Mandalam) Lunar zone
  Lunar Region, located at the center of two eye brows.

13. MALAM – 3 (THREE IMPURITIES OF THE SOUL):

- **AANAVAM :**

  This act makes clarity of thought, knowing the power of the soul, yielding to the Egocentric consciousness like 'I' and 'Mine' considering everything is to his own. (Greediness)

- **KANMAM :**

  Goes in collusion with the other two and responsible for incurring paavam (the Sin) and Punniyam (virtuous deed/Sanctity)

- **MAYAI :**

  Claiming ownership of the property of someone else and inviting troubles.

14. THODAM – 3 (THREE HUMOURS) :

- **VALI (VATHAM) :**

  It is a creative force, formed by Vaayu & Aakaya bootham.

- **AZHAL (PITHAM)**

  It is a protective force, formed by Thee bootham

- **IYYAM (KABAM)**

  It is a destructive force, formed by Mann & Neer bootham

15. EADANAI - 3 (PHYSICAL BINDINGS) :

Materialistic affinity Sibbling / Familial bonding

- **Porul patru -** Material bindings
- **Puthalvar patru -** Offspring bindings
- **Ulaga patru -** Worldly bindings

16. GUNAM – 3 (THREE COSMIC QUALITIES) :

- **Sathuva Gunam (*Characters of Renunciation or Ascetic Virtues*) :**

  The grace, control of sense, wisdom, penance, generosity, excellence, silence and truthfulness are the qualities attributed to the benevolent trait.

- **Raso Gunam (*Characters of Ruler*) :**

  Enthusiasm, wisdom, valour, virtue/penance offering gift, art of learning and listening are the 8 traits.

- **Thamo Gunam (*Carnal and Immoral Characters*) :**

  Immortality, lust, killing laziness, violation of justice, gluttony falsehood, forgetfulness and fraudulence etc.

17. VINAI – 2 (ACTS) :

- **Nalvinai -** Good Acts (Meritorious acts)
- **Theevinai -** Bad Acts (Sinful acts)

18. RAGAM – 8 (THE EIGHT PASSIONS) :

- Kaamam – Desire
- Kurotham – Hatred
- Ulobam – Stingy
- Moham – Lust (Intense or Sexual desire, infatuation)
- Matham – Pride (The feeling of respect towards one's self)
- Marcharyam – Internal conflict, Envy
- Idumbai – Mockery
- Ahankaram – Ego

19. AVATHAI – 5 (FIVE STATES OF CONSCIOUSNESS) :

- **NINAIVU-AWAKENED STATE *(Sakkiram)***

  This state exists between the eye-brows. The four strengths, the five senses, the five actions *(Asayam)* and the four *Andhakaranas* are active in this state.

- **KANAVU- Dream state *(Swappanam)***

  Dream state is one in which the five senses and five actions lie dormant at Adam's apple (Throat).

- **URAKKAM- Sleeping state *(Suzhuthi)***

This is the state in which the Anthakaranas are associated with the soul but these could not be expressed to others and its seat being thorax.

- **PERURAKKAM- Deep sleep *(Turiyam)***
  The seevathma, along with wisdom lies at the navel region, here respiration takes place.

- **UYIRPADAKKAM- Immersed state of seevathma *(Turiyatheetham)***
  The seevathma is deeply immersed in the moolathara without the awareness of impurity (malam), sloth (Mantham), delusion (maya) andother sense of touch.

THE UYIR THATHUKKAL :

The physiological units of the Human body are **Vali** (Vatham), **Azhal** (Pitham) and **Iyyam** (Kabam). They are also formed by the combination of the five elements.

Vaatham	**=**	**Vaayu +Aagayam**	**: Creative force**
Pitham	**=**	**Thee**	**: Force of preservation**
Kabam	**=**	**Mann +Neer**	**: Destructive force**

As per the above lines the Universe and the human body are made of five elements. If these three humours are in the ratio 1:½:¼ in equilibrium or in normal condition, then they are called as the Life forces.

SITES OF UYIR THATHUKKAL :

" பொங்கி ய வளந்துக்குள் பொல்லாதது இம் மூன்றான்
தங்கி ய வாயு சமத்தன்மனாவாதம்
பங்கி ய வன்னியால் டருந்தது பி த்தமே
டருந்த சலத்தி ல் பரி சி க்கும் நல்லையும்
வளந்த இம்மூன்றால் வளர்ந்தது நோயெல்லாம்
அந்தது தானி ன்று அவி பட்ட போகி கள்
மகி ழ்ந்தே யி தி ல் நி ன்ற மயக்கம் தி வதே"

- பதி னெண்சி த்தர் நாடி சாஸ்தி ரம்

THE FORMATION OF UYIR THATHUKKAL

மூலகை நாடியும் உயி ர் தாதுவும்

"தாது முறையே தன்இட வாதமாம்
போது பித்தலை கபந்து பித்தமாம்
மாது சுழுனைவழங்கி டும் ஐயமாம்
ஏது முற பார்த்து உரைந்தவர் சித்தரே"

- திருமூலர் சித்தர் நாடி சாஸ்திரம்

மூன்று வாயுவும் உயிர் தானும்

"உரைந்த ஆணல்லும் அந்த வாதத்தில்
பரைந்த பிராணன்படும் அந்தப் பித்தத்தில்
அரைந்த சமானன் அங்கும் கபத்தோடு
இணைந்தி வை மூன்றுக்கு ஒத்த குறி ஒன்றே"

- திருமூலர் சித்தர் நாடி சாஸ்திரம்

Vali	=	Abanan	+ Idagalai
Azhal	=	Piranan	+ Pinkalai
Iyyam	=	Samanan	+ Suzhumunai

I.VALI (VATHAM) :

a) THE NATURE OF VALI :

Vali is soft, fine and the temperature (coolness and hotness) could be felt by touch.

b) SITES OF VALI :

"தெளிந்திட்ட வாதமானதைப் பற்றி
நிறைந்தி லவைச் சேர்ந்துந்தி க்கீழே நின்று
குளிந்திட்ட மூலாதா டெழுந்து காமக்
கொடியி லவைப் பற்றி டெழுங் குதத்தைப் பாரே
நிணனொருத்தி டமும் ரோமக் காலும்
நிறைகி மாங்கி சமெல் லாம்பரந்து

- வைத்திய சதகம்

According to Vaithya sathakam, Vali dwells in the following places: They are Umbilicus, rectum, faecal matter, abdomen, anus, bones, hip joint, navel plexus, joints, hair follicle and muscles.

"அறிந்திடும் வாத மடங்கு மவத்தினில்"

- திருமூலர்

"நாமென்ற வாதத்துக் கிருப்பி டமே கேளாய்
நாபிக்குக் கீழென்று நவிலலாகும்"

- யூகி முனிவர்

According to Sage Thirumoolar and Yugi muni, the places of vatham are the anus and the region below the naval.

c) THE PROPERTIES OF VALI :

" ஏழுக்கு னேதாதேழ் மூச்சோங்கி இங்க
ஏழ்சி லெற எட்டினையுமாற்ற ஏழ்தி ரி ய
கேகம் டலைங்கு மேச் சறுசுறுப்பு
வகளிக்கும் மாந்தர்க்கு வாயு"

- சி த்த மருத்துவாங்க சுருக்கம்

d) THE FUNCTIONS OF VALI :

1. To stimulate the respiration
2. To activate the body, mind and the intellect.
3. To expel the fourteen different types of natural reflexes.
4. To activate seven physical constituents in functional co- ordination.
5. To strengthen the five sense organs.

In the above process vatham plays a vital role to assist the body functions.

II. AZHAL (PITHAM) :

a) THE NATURE OF AZHAL :

The nature of Azhal is atomic. It is sharp and hot. The ghee becomes watery, salt crystallizes and jaggery melts because of heat. The heat of Azhal is responsible for many actions and their reactions.

b) SITES OF AZHAL :

“தானனபி த்தம் பி ன்கலைவப் டற்றி ச்
சாய்வானபி ராணாயு தனைச் சேர்ந்து
னானநீ ர்ப்வயி ழறு மூத்
தநி த்தெறுந்த வக்கி னைவ யறவு செய்து
மாலேகே சிறுநத்தி லி ருப்பு மாகி
னோனனசி ரந்தனிலே யி றக்க மாகி
னொறி ற் பி த்தநி லை சறு னேமே”

- வைத்தி ய சதகம்

According to vaithiya sathagam, the pingalai, urinary bladder, stomach and heart are the places where Azhal sustains. In addition to the above places, the umbilicus, epigastric region, stomach, sweat, saliva, blood, essence of food, eyes and skin are also the places where Azhal sustains. Yugi muni says that the Azhal dwells in urine and the places below the neck.

c) THE CHARACTERS OF AZHAL :

Azhal is responsible for the digestion, vision, maintenance of the body temperature, hunger, thirst, taste etc. Its other functions include thought, knowledge, strength and softness.

d) THE FUNCTIONS OF AZHAL :

1. Maintenance of body temperature.
2. Produces reddish or yellowish colour of the body.
3. Produce heat energy on digestion of food.
4. Produces sweating.
5. Induces giddiness.
6. Produces blood and the excess blood are let out.
7. Gives yellowish coloration to the skin, eyes, faeces and urine
8. Produce anger, heat, burning sensation, inaction and determination.
9. Gives bitter or sour taste.

e) THE TYPES OF AZHAL :

- **Aakkanal – Anal pitham or Pasaka pitham – The fire of digestion.**

  It lies between the stomach and the intestine and causes digestion and dries up the moist ingested substance.

- **Vanna eri – Ranjaga pitham – Blood promoting fire.**
  The fire lies in the stomach and imparts red colour to the chyme and produces blood. It improves blood.
- **Aatralanki – Saathaga pitham – The fire of energy.**
  It gives energy to do the work.
- **Nokku Azhal – Alosaga pitham – The fire of Vision.**
  It lies in the eyes and causes the faculty of vision. It helps to visualize things.

- **Ul oli thee – Prasaka pitham – the fire of brightness.**
  It gives colour, complexion and brightness to the skin.

III. IYYAM (KABAM) :

THE NATURE OF IYYAM :

Greasy, cool, dull, viscous, soft and compact are the nature of Iyyam.

THE SITES OF IYYAM :

“சுரி னொஞ்சி தேமது சமானவய்வைக்
கொழு பே சழி முலையப் டற்றி விந்தி ல்
கீ றி பே சி ரசி லாக் கி லையச் சேர்ந்து
சி ங்குலவி ண்ணக்குரி ணமச்சை ரத்தம்
மீ றி பே நி றங்கோணரம் டெழும்பி ல்
மேவியதோர் மூலமிருங் குலி ற் கண்ணல்
தேறி யதோர் பொருத்தி டங்க ளெல்லாஞ்சேர்ந்து
சி தேமது வீற்றி ருக்குந் தி டங் கண்மே”

- வைத்தி ய சதகம்

Head, tongue, eyes, nose, throat, thorax, bone, bone marrow, joints, blood, fat, sperm and colon are the seats of Iyyam. It also lies in the stomach, spleen, the pancreas, chyle and lymph.

c) THE PROPERTIES OF IYYAM :

Stability, greasiness, formation of joints, the ability to withstand hunger, thirst, sorrow and distress are the qualities. It also helps to withstand sufferings.

d) THE FUNCTIONS OF IYYAM :

Greasiness, strength, roughness, knowledge, cool, growth, heaviness of bone, restriction of joint movements, pallor, indigestion, deep sleep and to have a sweet taste in tongue are the functions of Iyyam. The skin, eyes, faces and urine are white in colour due to the influence of Iyyam.

e) THE TYPES OF IYYAM :

- **Ali iyyam – Avalambagam:**
  Heart is the seat of Avalambagam. It controls all other types of Iyyam.

- **Neerpi iyyam – Kilethagam :**
  Its location is stomach. It adds moisture & gives softness to the ingested food.
- **Suvai kaan iyyam – Pothagam :**
  Its location is tongue. It is responsible for the sense of taste.
- **Niraivaiyyam – Tharpagam :**
  It gives coolness to the vision.
- **Ondri iyyam – Santhigam :**
  It gives lubrication to the bones particularly in the joints.

THE UDAL THATHUKKAL (PHYSICAL CONSTITUENTS) :

Udal Thathukkal is the basic physical constituents of the body. They are also constituted by the Five Elements.

1. Saaram	**:**	This gives mental and physical perseverance.
2. Senneer	**:**	Imparts colour to the body and nourishes the body.
3. Oon	**:**	It gives shape to the body according to the physical activity and cover the bone.
4. Kozhuppu	**:**	It lubricates the joints and other parts of the body to function smoothly.
5. Enbu	**:**	Supports the frame and responsible for the postures and movements of the body.
6. Moolai	**:**	It occupies the medulla of the bones and gives strength and softness to them.

7. Sukkilam/Suronitham : It is responsible for reproduction. These are the seven basic constituents that form the physical body. The bones are predominantly formed by the Earth component, but other elements are also present in it. All the three humours Vali, Azhal and Iyyam present in this 7 constituents. The intake food converted to udal thaadhu in which the intake food is converted to saaram in the first day, and then it converted to chenneer in the Second-day, oon, kozhuppu, enbu, moolai and sukkilam/ Suronitham respectively in the following days. So in the seventh day only the intake food goes to the sukkilam/suronitham.

UDAL THEE (FOUR KINDS OF BODY FIRE) :

There are four kinds of body fire. They are Samaakkini, Vishamaakkini, Deekshaakkini and Manthaakkini.

- **SAMAAKKINI (BALANCED DIGESTIVE FIRE) :**
  The digestive fire is called as Samaakkini. This is constituted by Samana Vayu, Anala Pitham and Kilethaga Kabam. If they are in normal proportion then it is called as Samakkini. It is responsible for the normal digestion of the food.

- **VISHAMAAKKINI (TOXIC DIGESTION) :**
  Due to deranged and displaced Samana Vayu, it takes a longer time for digestion of normal food. It is responsible for the indigestion due to slow digestion.

- **DEEKSHAAKINI (ACCENTUATED DIGESTION) :**
  The samana vayu rounds up the Azhal, which leads to increased Anala Pitham, so food is digested faster.

- **MANTHAAKKINI (SLUGGISH DIGESTION) :**
  The samana vayu rounds up the Iyyam, which leads to increased Kilethaga Kapham. Therefore food is poorly digested for a very longer period and leads to abdominal pain, distension heaviness of the body etc.

THINAI :

There are five thinai (The Land)

- **Kurinchi** - Mountain and its surrounding areas (Hilly terrain)
- **Mullai** - Forest and its surrounding areas (Forest ranges)
- **Marudham** - Agricultural land and its surrounding areas (Cultivable lands)
- **Neidhal** - The coastal and its surrounding areas (Coastal belts)
- **Paalai** - Desert and its surrounding areas (Arid Zone)

FEATURES OF THE FIVE REGIONS :

1. KURINCHI :

"குரி ஞ்சி வரி ஷ்தி ற்கு கொற்றமுல் ரத்தம்
உரி ஞ்சி வரைமுழ்மம் - அரி ருரைக்
கையோ தங்குரா தாலைல்லை யங்கதி க்கும்
ஐயோ தங்கும் அரி "

- டதார்த்த குணசி ந்தாமணி

Fever causing anemia, any abnormal enlargement in the abdominal organ (vaitrulaamai katti) also leads to Iyya disease.

2. MULLAI :

"முல்லை நி லத்தியோ ழுரி நி ரை மேவி னுலவ்
வெல்லை நி லைத்தி த்த மெய்தாங்காண் வல்லையின்
வாதமொழி யாதனுள் மன்னுலையோ நோய்ப்
பேதமொழி யாதறையப் பி ன்பு"

- டதார்த்த குணசி ந்தாமணி

This mullai land leads to Azhal, Vallai & Vali diseases.

3. MARUDHAM :

"மருதி லம் நன்னீர் வளமாலைக் கொண்டீ
பொருனில மாதி யதோய் போக்கும் - கருதி த்
தாறி ரதஞ்சூ ழந்தமிரன்றாற்பி ணிவெல்
லோரி ரதஞ்சூழ்வி க்குமி ல்"

- டதார்த்த குணசி ந்தாமணி

All the Vali, Azhal and Iyyam disease will be cured in this land.

4. NEIDHAL :

"நெய்தனில மேலுவை நீ ங்கா தரி னுது
வெய்தனில மேதங்குவீ டாகும் - நெய்தல்
மருங்குலை மி க்காக்கும் வல்லுவை வீ க்கும்
கருங்குலைக் கீ ழிறக்கு காணீ

- டதார்த்த குணசி ந்தாமணி

This place induces Vali diseases and affects liver and intestines.

5. PAALAI :

"பாலை நி லம்போற் படரைப் பி றப்பி க்க

மேனி லமி யாதுவி ரி த்தற்கு- மேலை நி ல
மும்பி னிக்கும் மி ல்லம் முறையே யவற்றகலம்
எப்பி னிக்கு மி ல்லை தென்

- பதார்த்த குணசி ந்தாமணி

This land produces all the three Vali, Azhal and Iyyam disease.

KAALAM :

Ancient Tamilians had divisions over the year into different seasons know as Perumpozhudhu and likewise in the day, it is known as Sirupozhudhu.

a. PERUMPOZHUDHU :

The year is divided into six seasons. They are,

- Kaarkalam – Aavani, Purataasi (August 16-October 15)
- Koothir – Aipasi, Kaarthigai (October 16-December 15)
- Munpani – Maargazhi, Thai (December 16-February 15)
- Pin pani – Maasi, Panguni (February 16-April 15)
- Ilavenil – Chithirai, Vaigaasi (April 16-June 15)
- Mudhuvenil – Aani, Aadi (June 16 – August 15)

b. SIRUPOZHUDHU :

The day has been divided into six parts of four hours each. They are maalai (evening), yammam (Midnight), Vaigarai (Dawn), Kaalai (Morning), Nannpakal (Noon), Erpaddu (Afternoon). The each perum pozhuthu and sirupozhuthu is associated with the three humours naturaly.

Table:1-POZHUTHUGAL

NILAM	POZHUTHU	
	PERUMPOZHUTHU	SIRUPOZHUTHU
Kurinchi	Koothir kaalam, Munpani	Naduiravu
Mullai	Kaarkaalam	Maalai
Marutham	Ilavenil, Venil, kaarkaalam, koothirkaalam, Munpani, Pinpani	Vaigarai, kaalai
Neidhal	Ilavenil, Venil, kaarkaalam, koothirkaalam, Munpani, Pinpani	Pirpagal
Paalai	Venil, Pinpani	Nadupagal

FOURTEEN NATURAL REFLEXES / URGES :

The natural reflexes excretory, protective and preventive mechanisms are responsible for the reflexes, urges and instincts. They are 14 in number

1. Vatham (Flatus)
2. Thummal (Sneezing)
3. Siruneer (Micturition)
4. Malam (Defecation)
5. Kottavi (Act of yawning)
6. Pasi (Sensation of hunger)
7. Neer vetkai (Sensation of thirst)
8. Erumal (Coughing)
9. Elaipu (Fatigue)
10. Thookam (Sleep)
11. Vaanthi (Vomiting)
12. Kanneer (Tears)
13. Sukkilam (Semen)
14. Suvasam (Breathing)

These natural reflexes are said to be an indication of normal functioning of our body. A proper maintenance should be carried out and they should not be restrained with force.

3.B. SIDDHA PATHOLOGY

KUGARANA NILAI IN SIDDHA MEDICINE

This is the first medical system to emphasis health as the perfect state of physical, psychological, social and spiritual components of human being. The condition of the human body in which the dietary habits, daily activities and the environmental factors influence to keep the three humors in equilibrium is considered as healthy living.

DISEASE

Disease is also known by other names viz sickness, distemper, suffering and ailment, distress of mind, chronic disease and dreadful illness.

1. THE CHARECTERISTIC FEATURES OF THE DISEASE

Diseases are of two kinds

i. Pertaining to the body

ii. Pertaining to the mind according to the variation of the three humors.

CAUSES OF DISEASE

Excepting the disease caused by our previous births, the disease is normally caused by our food habits and actions.

This has been rightly quoted in the following verses by Sage Thiruvalluvar,

"மி கி ஹ் குறை ஹ் நோய்செய்யும் நூலோர்
வி முதல் எண்ண ழுது"

- தி குள்ளர்

The food and actions of a person should be in harmony with the nature of his body. Any increase or decrease in a humor viz. Vatham, Pitham, Kabam leads to the derangement of the three humors. The acceptance of food means the taste and quality of the food eaten and a person's ability to digest. 'Actions' mean his good words, deeds or bad actions. According to Sage Thiruvalluvar, the disease is caused due to the increase or decrease of three humors causing the upset of equilibrium. So disease is a condition in which there is derangement in the five elements, which alters the three humors, reflected in turn in the seven physical constituents. The change could be an increase or decrease in the humors. This shows the following signs as per vitiation of the individual humor.

2. QUANTITATIVE CHANGES OF UYIR THATHUKKAL

HUMOUR	INCREASED	DECREASED
VALI (Vatham)	Wasting, blackish discoloration, affinity to hot foods, tremors, distended abdomen, constipation, weakness, insomnia, weakness in sense organs, giddiness and laziness.	Body pain, feeble voice, and diminished capability of the brain, decreased intellectual quotience, syncope and increased kaba condition.
AZHAL (Pitham)	Yellowish discoloration of conjunctiva, skin, urine and feces, polyphagia, polydypsia, dyspepsia, burning sensation all over the body and decreased sleep.	Loss of appetite, cold, pallor and features of increased kabam.
IYYAM (Kabam)	Loss of appetite, excessive salivation, diminished activity, heaviness, pallor, cold, decreased physical constituents, dyspnoea, flatulence, cough and excessive sleep.	Giddiness, dryness of the joints and prominence of bones. Profuse sweating in the hair follicles and palpitation.

Table-2- Changes of Uyir Thathukkal

3. UDAL THATHUKKAL

UDAL THATHUKKAL	INCREASED FEATURES	DECREASED FEATURES
SAARAM	Loss of appetite, excessive salivation, diminished activity, heaviness, pallor, cold, decreased physical constituents, dyspnoea, flatulence, cough and excessive Sleep	Dryness of skin, tiredness, loss of weight, lassitude and Irritability while hearing louder sounds.
SENNEER	Boils in different parts of the body, splenomegaly, tumours, pricking pain, loss of appetite, haematuria, hypertension, reddish eye and skin, leprosy and jaundice.	Affinity to sour and cold food, nervous debility, dryness and Pallor.
OON	Tubercular adenitis, venereal diseases, extra growth around neck, cheeks, abdomen, thigh and genitalia.	Lethargic sensation, pain in joints, muscle wasting in mandibular region, gluteal region, penis and thighs.
KOZHUPPU	Feature of increased musculature, tiredness, dyspnoea on exertion, extra musculature in gluteal region, external genitalia, chest, abdomen and thighs.	Loins pain, splenomegaly and emaciation.
ENBU	Excessive ossification and redundant dentition	Joint pain, falling of teeth, falling and splitting of hairs and nails.
MOOLAI	Heaviness of the body and eyes, Swollen Inter phalangeal joints, oliguria and non-healing ulcers	Osteoporosis and Blurred vision.
SUKKILAM (OR) SURONITHAM	Increased sexual activity, urinary calculi	Dribbling of sukkilam / suronitham or senneer during coitus, pricking pain in the testis and inflammed& contused external genitalia.

Table-3-Changes of Udal Thathukkal

4. TASTE

TASTES	DISEASES DUE TO HIGH INTAKE

Inippu	Develops obesity, excessive fat, increased mucous secretion, indigestion, diabetes, cervical adenitis, increased kabam and its diseases
Pulippu	Develops nervous weakness, dull vision, giddiness, aneamia, dropsy, dryness of tongue, acne, blisters etc.
Uppu	Ageing, hair loss, leprosy, dryness of tongue, debility
Kaippu	Increased dryness of tongue, defective Spermatogenesis, body weakness, dyspnoea lassitude, tremor, back and hip pain
Kaarppu	Dryness of tongue, generalized malaise, tremor, back pain, lassitude etc.
Thuvarppu	Abdominal discomfort, chest pain, tiredness, impotency, vascular constriction, constipation, dryness of tongue etc.

Table-4-Taste

5.KAALAM

KAALAM (Season)	KUTTRAM	STATE OF KUTTRAM
1. Kaarkaalam (Rainy) Aavani –Puratasi (Aug 16 – Oct 15)	Vatham ↑↑ Pitham ↑ Kabam (--)	Ectopic escalation In situ escalation Restitution
2. KoothirKaalam **(Post rainy)**Iypasi –Karthigai (Oct 16 – Dec 15)	Vatham (--) Pitham ↑ ↑ Kabam (--)	Restitution Ectopic escalation Restitution
3. MunpaniKaalam (Winter) Markazhi – Thai (Dec 16 – Feb 15	Vatham (--) Pitham (--) Kabam ↑	Restitution Restitution Restitution
4. PinpaniKaalam **(Post winter)** Masi – Panguni (Feb 16 –Apr 15)	Vatham (--) Pitham (--) Kabam ↑ ↑	Restitution Restitution In situ escalation
5. IlavenilKaalam (Summer) Chithirai – Vaikasi (Apr 16 – Jun 15)	Vatham (--) Pitham (--) Kabam ↑ ↑	Restitution Restitution Ectopic escalation
6. MudhuvenilKaalam **(Post summer)** Aani – Aadi (Jun 16 – Aug 15)	Vatham ↑ Kabam (--)	In situ escalation Restitution

Table 5.changes in climatory condition of the external world has its corresponding effects on the human organs

6.THINAI

THINAI	LAND	HUMOURS

1. Kurinchi	Mountain and its surroundings - Hilly terrain	Kabam
2. Mullai	Forest and its surroundings - Forest ranges	Pitham
3. Marutham	Farm land and its surroundings - Cultivable lands	All three humors are in Equilibrium
4. Neidhal	Sea shore and its adjoining Areas-Coastal belt	Vadham
5. Paalai	Desert and its surroundings- Arid zone	All three humors are affected

Table-6-Thinai, Land, Humours

ALTERATION IN REFLEXES (14 Vegangal)

There are 14 natural reflexes involved in the physiology of normal human being. If wilfully restrained or suppressed, the following are resulted.

- **Vatham (Flatus)**

This urge should not be suppressed. If it is suppressed it leads to chest pain, epigastric pain, abdominal pain, aches, constipation, dysuria and indigestion predominate.

- **Thummal (Sneezing)**

If restrained, it leads to headache, facial pain, low back pain and neurotic pain in the sense organs.

- **Siruneer (Urine)**

If restrained, it leads to urinary retention, urethral ulcer, joint pain, pain in the penis, gas formation in abdomen.

- **Malam (Feces)**

If restrained, it leads to pain in the knee joints, headache, general weakness, flatulence and other diseases may also originate.

- **Kottavi (Yawning)**

If restrained, it leads to indigestion, leucorrhoea, and abdominal disorders.

- **Pasi (Hunger)**

If restrained, it leads to the tiredness of all organs, emaciation, syncope, apatheticface and joint pain.

- **Neervetkai (Thirst)**

If restrained, it leads to the affection of all organs and pain may supervene.

- **Kaasam (Cough)**

If it is restrained, severe cough, bad breath and heart diseases will be resulted.

- **Ilaippu (Exhaustiveness)**

If restrained, it will lead to fainting, urinary disorders and rigor.

- **Nithirai (Sleep)**

All organs will get rest only during sleep. So it should not be avoided. Disturbance will lead to headache, pain in the eyes, deafness and slurred speech.

- **Vaanthi (Vomiting)**

If restrained, it leads to itching , anaemia, eye diseases and symptoms of increased Pitham.

- **Kanneer (Tears)**

If it is restrained, it will lead to Sinusitis, heart diseases, headache, eye diseases.

- **Sukkilam (Semen)**

If it is restrained, there will be joint pain, difficulty in urination, fever and chest pain.

- **Suvasam (Breathing)**

If it is restrained, there will be cough, abdominal discomfort and Anorexia.

3.C. DIAGNOSTIC METHODOLOGY

The methodology of diagnosing disease in Siddha system shows uniqueness in its principle. The principle comprises of examination of Tongue, Complexion, Modulation in speech, inspection of eyes and findings by palpation. It also includes examination of urine and stool. The reinforcement of Diagnosis is based on Naadi (Pulse) examination. All these together constitute 'Envagai thervugal' which forms the basis of diagnostic methodology in Siddha system of Medicine.

These tools not only help in diagnosis but also to observe the prognosis of the disease and for reassuring the patient and to be informed about the nature of diseases. Besides these Envagaithervugal there are some other parameters in Siddha system which are greatly helpful in diagnosing various disease, they are Madikadainool (Wrist circummetric sign) and Soditham (Astrology).

ENVAGAI THERVUGAL (Eight fold examination)

The eight such diagnostic methods, collectively referred to as "Envagai thervu (Eight type) Thervugal(Examination)" in Siddha system.

"அத்து றோவை கரத்தாம வைம்போல்
படுத்தரி வீர் நாடிப் பரி சம் - தொகுத்த நி றம்
கட்டுரைச் சொல்மொழி க் கண் மல மூத்தி ரம் நா
எட்டுவகை யாலுமறி வீர்"

- அத்தி யர் வைத்தி ய சி ந்தாமணி 4000

Various aspects of Siddha regarding 'Envagai Thervu'

"நாடி பரி சம் நாநி றம் மொழி விழி
மலம் மூத்தி ரமி வை மருத்துரையாதம்"

- தேரையர்.

"மெய்குறி நி றந்தொனி விழி நா வினுலம் வைக்குறி "

- தேரையர்

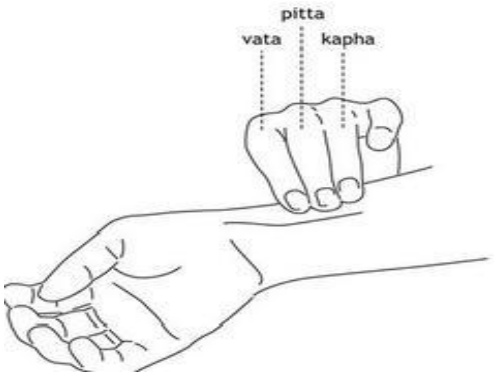
The eight methods of diagnosis are Naadi (Pulse), Sparisam (Palpation), Naa (Tongue), Niram (Color), Mozhi (Voice), Vizhi (Eyes), Malam (Feces) and Neer (Urine).

1. NAADI (Examination of pulse)

The pulse Diagnosis is a unique method in Siddha Medicine. The pulse should be examined in the right hand for male and the left hand for female. The pulse can be recorded at

the radial artery. By keenly observing the pulsation, the diagnosis of disease as well as its prognosis can be assessed clearly.

Naadi is nothing but the manifestation of the vital energy that sustains the life with in our body. Naadi plays an important role in Envagaithervu and it has to be considered as foremost thing in assessing the prognosis and diagnosis of various diseases. Any variation that occurs in the three humors is reflected in the Naadi. These three humors organize, regularize and integrate basic functions of the human body. So, Naadi serves as good indicator of all ailments.



நாடி பார்க்கும் வகை

"இயல்நற் நாடிகள் பார்க்கும் வகைகேளு
என்னென்றால் நடுவிரல் நீ விட்டி ன்னே
அயல்நற் அந்தவிரல் மோதி ரமாம் விரலை
அடனேஇந்தி வ்பு சுண்டுவிரலி ஞ்து
உயல்நற் தண்டுவிரலி ஞ்து அப்பால்
உத்தமொரு அங்குட விரலைநீ விக்கரத்தி ல்
டயல்நற் சீ யோதி அங்குல மோதன்னி
டார்தவிட மூன்றாம் சரம்பார்க்கும் வகையே
வகைன்னவாதமதுவுண்டையாம் பி த்தம்
வலையொன்று அங்கால் வலையாய்தி ற்கி ல்
டலையில்லை நாடிகளு தொந்த மி ல்லை
டன்னான்சகுரோசருநக் கூடுசொன்னேன்"

- அத்தி யர் கலைமன 100

Naadi is felt by

Vali - Tip of index finger

Azhal - Tip of middle finger

Iyyam - Tip of ring finger

மூன்றையும் மாத்திரை அளவும்

"வழங்கிய வாதம்மாத்திரை ஒன்றாகில்
வழங்கிய பித்தம் தன்னில் அரைவசி
ஆங்கும் கபந்தானுங்கி லோகோடில்
பிழங்கிய சீர்க்குப் பிசகொன்று மில்லையே"

- குணவாகட நாடி

The pulse is measured in wheat/grain expansile heights. The normal unit of pulse diagnosis is 1 for Vali (Vaadham), ½ for Azhal (Pitham) and ¼ for Iyyam (Kabam).

நாடி நடை

"வாகி லங்கோழி மயிலனைநடக்கும் வாதம்
ஏகிய வரையட்டை யெலியனைநடக்கும் பித்தம்
போகிய தவளைபாம்பு போலவும் சேத்துமந்தான்"

- குணநாடி

Compared to the gait of various animals, reptiles and birds.

Vali	-	Gait of Swan and peacock
Azhal	-	Movement of Tortoise and Leech
Iyyam	-	Leaping of Frog and crawling of a Serpent

2. SPARISAM (Examination by touch)

TOUCH (தொடு உணர்வு):

"வெம்மை குறைந்தாலுமிகுத்தாலும் வாதபித்தம்
தம்மை நிரைநிரையாய்ச் சாற்றுவர்- வெம்மைமிகு
சீதமுறுவராகில் சிலேட்டுமமொன்றுதொந்த
மீதமுறுவராகுமேல்"

- அத்தியர் வைத்தியசிந்தாமணி 4000

"நேமமுனைவாதத்தின்றேசந்தானும்
நேர்மையாய்குளிர்ந்துசிலவிடத்திலேதான்
மாமமுடலுறும் தூதுபட்பு
மாதலாம் பித்தத்தின்தெசந் தானும்
தோயவேவுணர்வாயிருக்குந் தெளிவாய்
சேத்துமத்தின்தேகமதுகுளிர்ந்திருக்கும்
பாயதொந்த தேகமதுபலவாறாகும்
பரிந்துதொட்டுத் தேகத்தைப் பார்த்துப்பேசே"

- கண்ணுமி பரம்பரை வைத்தியம்

In Vali disease, some regions of the body felt chill and in some areas they are hot.

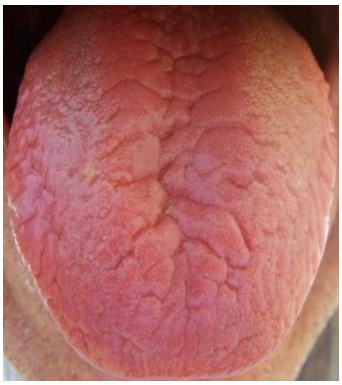
In Azhal disease, we can feel heat.

In Iyya disease, chillness can be felt.

In Thontham diseases, we can feel altered sensations.

3. NAA (Examination of tongue)

VATHAM PITHAM KABAM



"மானசுரி யரி யும் நாவின்சுற்றை
மகர்கி ரோன்வதரோகி யிற்றன்நாவு
கமாக வெடித்து கறுத்தி ருக்கு முடோல்
கரு கொய்மை பித்தரோகி யிற்றன்நாவு
நமை சி வந்து டச்சென்றி ருக்கும் நட்பி ன
சி வேத்துரோகி யிற்றன்நாவு
தமதென்றுதி போர்கள் சொன்ன
தன்மையடி தடித்து வெந்தி ருக்கும் பாரே"

- கண்ணுமி பரம்பரை வைத்தியம்

In Vali derangement, tongue will be cold, rough, furrowed and tastes pungent.

In Azhal, it will be red or yellow and bitter taste will be sensed.

In Iyyam, it is pale, sticky and with lingering of sweet taste.

In Thontham, tongue will be dark with raised papillae and dryness.

4.NIRAM (Examination of complexion)

"தேகத்தி னிறந்தானுஞ்செட்டக் கேளர்
சி றுமையாய் வதந்தான்கறுத்தி ருக்கும்
போகத்தி ன்பித்தரி றமஞ் ளாகும்
பெருஞ்சேத்ம ரோகி க்கு வெளுப் தாகும்

பாகத்தி ன்தொந்தரோ கி க்குத் தாலும்
டலமைன்னோகி ப் பரந்து நி ற்கும்'

- சி த்த மருத்துவாங்கச் சுருக்கம்

In Vali, Azhal and Iyyam variations, the colour of the body will be dark, yellow or red and fair respectively.

"உரத்தகற் பான்வாத ரோகி பி த்த ரோகி
அரத்தமஞ் னைக்குரித்தோன் ஆன்- இத்தம்
குரித்தனுமாவான்கொடுஞ்சி லேத்தும ரோகி
வெஞ்சி டுவான்தொந்த ரோகி பே'

- அத்தி யர் வைத்தி ய சி ந்தாமணி- 4000

According to Agathiyar Vaithiya Chinthamani Venba – 4000, In Vatha ,Pitha and Kaba vitiations the colors of body like as yellow, red and pale.

"மூன்றாகும் வாதபி த்த சி லேத்துமத்தால்
மி குந்தமுதொந்தி த்த ரோகி தேகம்
தோன்றாத சீ தய வெஞ்சு காலமூன்றும்
தொகுத்தேவான்தி ரேகத்தி நி றத்தைக் கேளு
வன்றாத வாதவுல் கறுத்துக் காணும்
மதி யபி த்த முல் சி வப்புப் டலைகானும்
போன்றாத வையவுல் வெண்மை தோன்றும்
பொருந்துந்தொந்த ரோகவுற் கி ஷ்ணை பொக்கும்'

- கண்ணுசாமி பரம்பரை வைத்தி யம்

According to Kannusamy Paramparai Vaithiyam, In Vatha, Pitha and kaba vitiations, the colors of the body like as black, reddish green and white. In Thontha constitution, the color of the body will be associated with combination of two humours.

"டலைவாத தேகநி றங் கறுத்து நி ற்கும்
வைத்தி யதேக நி றமஞ்சள் சி வப்தாமே
தாமே சி லேட்டு மதேகநி றம் வெளுப்பு தான்
தொந்தேகம் இந்நால் வி தமாய நி ற்கும்'

- தன்வந்தி ரி (பதி னெண்சி த்தர் நாடி சாத்தி ரம்)

According to Pathinen Siddhar Naadi Nool, In Vatha, Pitha and Kaba vitiations, the colors of the body like as black, yellowish red and white. In Thontha constitution, the color of the body will be associated with combination of two humours.

5.VIZHI (Examination of Eyes)

"வண்மையாய் கண்ணுறுதி ப்படைத்தேள்வதம்
உற்றவிழி கறுத்துநொந்து நீருங் காணும்
தண்மையாப் பித்தரோகி யின்கண்கள்
சார்பாகப் படலமிவ் வேறுங் காணும்
வெண்மையா வையரோகி விழிகள்தானும்
வானவெண்மை றதோ நாதம்
திண்மையாத் தொந்தரோகி யின்கண்கள்
தீட்டுமாய் படிறமென்றறைய லாமே"

-கண்ணுறுமி பரம்பரை வைத்தியம்

"காணுறுவ தரோகிக்கு கண்கள்
கருநிறமாய் தொந்தமுக தண்ணீர்பாயும்
பூணுற்ற பித்தரோகி கடிமஞ்சள்போலிருக்கம்
சிவப்புநிறப்போலுவதோன்றும்"

-பதினெண்சித்தர் நாடி சாத்திரம்

In Vali disease the tears are darkened.

In Azhal disease tears are yellow.

In Iyya disease tears are whitish in colour

In Thontha disease the tears are multi tinged.

In Vali disease there will be excessive tears (epiphora).

In disturbance of all three humors, eyes will be inflamed and reddish.

6.MOZHI (Examination of voice)

"பார்படான்வதரோகி யின்வார்த்தை
படகுமாய்ச் சமசத்த மாயிருக்கும்
சேர்ப்படதான்பித்தரோகி யின்வார்த்தை
செப்பக்கோளலெத்துமே யுறத்திருக்கும்
ஏற்படதான்ஐயரோகி யின்வார்த்தை
மெள்ளதாகச் சிறுத்திருக்கியம்பி தாகும்
கோற்கவேயிம்மூன்றுந் தொந்தமாகில்
சமமாற் படிதமாய் கோவரே"

-கண்ணுறுமி பரம்பரை வைத்தியம்

In variation of Vali, Azhal and Iyyam the voice will be medium, high and shrill/low pitched respectively. By the voice, the strength of the body can be assessed.

7.MALAM (Examination of feces)

"ஒக்குமே வாத நோய் மத்தைப் பார்க்கில்
உகந்தமஞ் சாறில் பெ காறுத்தி ருக்கும்
மிக்கபித்த நோய்மத்தை யுற்றுப் பார்க்கில்
மிகுந்தசி வப்புன்சலை தானுந் தோற்றும்
வைக்குளை மாலேகை வாய் ரோகம்
மயதான்வெண்மைநிற மாயி ருக்கும்
பக்குமா யிம்முந்நூ தொந்தி ப் பாகில்
பகருமின்ன நிறங்களை பரி ந்து காணும்"

- கண்ணுமி பரம்பரை வைத்தி யம்

In exacerbated Vali, faces is hard, dry and darker .

In Azhal vitiation, it is yellow.

In Iyyam disturbances it is pale

In Thondham, it is a mixture of all colours.

8. MOOTHIRAM (Examination of urine)

“ஓங்கி யவாதத்தோர்க்கு நீர்வெளுத்துக் குன்றா நுரைக்கிஞ்
சூங்கொடி கடுத்து நொந்து சிறுத்துன்பொருமி விழும்
பாங்குன்பித்தத்தோர்க்குப் பசியநீர் சிவந்து காட்டி
ஏங்கவே கறுக்கதாக எரித்துன்கடுத்து வீழும்
வீழுமே சிலேற்மத்தோர் நீர்க்குமே வெளுக்கக் கோய்
நாழுமே வெளுத்தறைந்து நலம்பெறவீழுங் கண்ணாய்
வாழ்வழி மானதொந்த ரோகமானிடர்க்குந் தானே
தாழுநீர் டலறிந்தா னகஸ்தியன் சொற்றி னேனே”

- கண்ணுசாமி பரம்பரை வைத்தியம்

For patients suffering from vatha diseases, the urine will be scanty and dysuria. For patiets suffering from pitham the urine will be greenish red in colour and there will be burning micturition.

தேரையர் நீர்க்குறி நெய்க்குறி

" ஆதுமாறி ரதமும் அதிரோதமதாய்
மீதல் அர்தல் அலைந்தவிர்ந்தழற்
குற்றமொந்தி உறங்கி வைறை
ஆத்மசத் தாவிபே காதுபெய்
தொருதுளித்தக் கலைக்குட்டு நீரின்
நிறக்குறி நெய்க்குறி நிறுமித்தல் கடனே

- தேரையர் நீர்க்குறி நெய்க்குறி

Theraiyar, one of the renowned authors of Siddha medicine described urine examination and stages of health. He had explained about the colour and consistency of the urine in vitiated humor and disease (Neerkuri). He also emphasized about the spreading nature of a single drop of oil on the surface of the urine indicating the imbalance of specific dosha and prognosis of disease (Neikkuri).

Neerkuri:

“வந்த நீர்க்குறி வகை மணம் நுரை எண்ணெய்

ஸ்ந்தி யழுகலை யறைது முறவே'

- தேரையர் நீ ர்க்குறி நெய்க்குறி

Five characters of urine have to be examined. They are colour, consistency, odour, frothy and deposits.

Colour of the urine

Normal urine is straw yellow coloured and mildly aromatic. The time of the day and food taken will have an impact on the colour of the urine.

Colour of the urine in diseased condition

Yellow colour (Similar to straw soaked water) - Indigestion
Lemon colour - Good digestion
Reddish yellow - Heat in body
Colour similar to flame of forest red or flame coloured - Excessive heat
Colour of saffron - Extreme heat

Neikkuri:

" அனைநீ ஸ்ளீதே வதம்
ஆ போல் பரவின் ஆதே பித்தம்
முத்தொத்து நி ற்கி ன்மொழி தென்கபமே'

- தேரையர் நீ ர்க்குறி நெய்க்குறி

The spreading pattern of oil drop is the indicative of Vali, Azhal and Iyyam diseases.
Aravu (Snake Pattern of spread) indicates Vali disease,
Aazhi (Ring Pattern of spread) indicates Azhal disease.
Muthu (Pearl Pattern of spread) indicates Iyya disease.
In Neikkuri, the rapid spread of oil drop; Pearl beaded and Sieve type of spreading pattern indicates incurable state of the disease. From this, we can assess the prognosis by the Neikkuri.

Indications of spreading pattern of oil

Lengthening - Vali
Splits - Azhal

Sieve - Iyyam

Stands as a drop - Poor prognosis

Slowly spreads - Good prognosis

Drop immerses into Urine - Incurable disease

MANIKKADAI NOOL(Wrist circumetric sign)

Ref:Agathiyar Soodamanikayaru Soothiram

"கமலக்க மணிக்கையி ல் கயறு சூத்தி ரம்
விமலனேநோக்கி பே யேமாமுன
தி மி லாம் பி னைது சேரச் செப்பி பே
அனைமுனிக்கு முன்னுரைச் செய்தே"

- பதி னெண்சி த்தர் நாடிநூல்

According to the Pathinen Siddhar Naadinool, Manikadainool is also helpful in diagnosis. This manikkadainool is a parameter to access the disease by measuring the circumference of the wrist by means of a thread and then expressing it in terms of patient's finger breadths. By this measurement the disease can be diagnosed.

Manikadai nool inference

(Ref: Agathiyar soodamanikayaru Soothiram)

When the Manikkadainool is 11 fbs, the person will be stout and he will live a healthy life for many years. When the Manikkadainool measures between 4 & 6, it indicates poor prognosis of disease and the severity of the illness will be high and it leads to death.

Measurement Possible conditions

- 10 fbs Pricking pain in chest and limbs, gastritis and ulcer result.
- 9 ¾ fbs Fissure, dryness and cough will be resulted.
- 9 ½ fbs Odema, increased body heat, burning sensation of eye, fever, Mega noi& Anorexia.
- 9 ¼ fbs Dysuria, Insomnia, Sinusitis and Burning sensation of Eye.
- 9 fbs Impaired hearing, pain around waist, thigh pain, unable to walk.
- 8 ¾ fbs Increased body heat, skin disease due to toxins, abdominal discomfort, cataract, sinusitis.
- 8 ½ fbs Leucorrhoea, venereal disorder and Infertility will occur.
- 8 ¼ fbs Stout and painful body. Headache, Sinusitis and toxins induced Cough.
- 8 fbs Abdominal discomfort, gastritis, anorexia & venereal diseases.
- 7 ¾ fbs Piles, burning sensation of limbs, headache, numbness occur.
- Within 2 years cervical adenitis and epistaxis results.
- 7 ½ fbs Osteoporosis, abdominal discomfort, burning sensation of eyes, increased body temperature. Within 6 days all the joints of the limbs presents a swelling.
- 7 ¼ fbs Lumbar pain, increased pitha in head, anemia, eye pain, odema and somnolence
- 7 fbs Pitham ascends to head, haemetemesis, phlegm, burning sensation of limbs and constipation.
- 6 ¾ fbs Eye ache, dizziness, testis disorder. Within 3 years it causes anuria, pain and burning sensation over limbs, facial sweating results.
- 6 ½ fbs Thirst, anorexia, increased body heat and vatham results.
- 6 ¼ fbs Diarrhea, belching, vomiting and mucous dysentery
- 6 fbs Reduced weight, phlegm in chest. It results in death within 20days.
- 5 ¾ fbs Delirium, dizziness, loss of consciousness. It results in death even if the patient takes gruel diet
- 5 ½ fbs Severity of illness is increased. Toxins spread to the head.Tooth darkens. Patient will die in 10 days.
- 5 ¼ fbs Patient seems to be sleepy and death results on the next day.

- 5 fbs Pallor and dryness of the body. Kabam engorges the throat and the person will die.
- 4 ¾ fbs Dryness of tongue and tremor present. Patient will die in 7days.
- 4 ½ fbs Shrunken eyes, odema will present and death results in 9 days.
- 4 ¼ fbs Tremor, weakness of limbs and darkening of face occurs.
- 4 fbs Pedal oedema will be present. Patient will die in 5 days.

Proteinuria is the presence of excess proteins in the urine. In healthy persons, urine contains very little protein; an excess is suggestive of illness. Excess protein in the urine often causes the urine to become foamy.

There are three main mechanisms to cause proteinuria:

- Due to disease in the glomerulus, because of increased quantity of proteins in serum (overflow proteinuria) and due to low reabsorption at proximal tubule.
- Proteinuria may be a sign of renal (kidney) damage. Since serum proteins are readily reabsorbed from urine, the presence of excess protein indicates either an insufficiency of absorption or impaired filtration. People with diabetes may have damaged nephrons and develop proteinuria. The most common cause of proteinuria is diabetes, and in any person with proteinuria and diabetes, the cause of the underlying proteinuria should be separated into two categories: diabetic proteinuria versus the field.

It is stated that in proteinuria urine will be foamy such a condition is explained in Theraiyar Neerkkuri Vaidyam:

**"பந்தமெய்ப் பசையு ணெட்டும் பருத்
தந்தர்ப் பூதமாய் நிலை மூத்தி ரத்தி ல்
சம்பந்தப்டும் ததி நுரைப் படலே'**
- தேரையர் நீ ர்க்குறி வைத்தி யம்

Urine seems to be frothy when the mei pasai that is kabam humour and its types gets deranged and comes out through urine along with vatham. When frothy urine that is Albuminuria in diabetes is considered the main causative factor for the disease that is the prime cause for Megam is pitham humour, initially it will get deranged, derangement of pitham will lead to mobilization of kabam from its original dwelling sites; this is stated as **"பந்தமெய்ப் பசையு ணெட்டும்"** and it accompanies vatha and comes out through urine and the urine will eventually appear frothy. Madhumegham come under Megha Roga Nidanam in Siddha , in certain types of Megha Rogam it is states that urine will be frothy.

மேக ரோக நி தானம்

தெளிவாக தேவி தான்சி வனநோக்கி த்
தெண்டீட்டு ஆதித்தத்தை முன்னே
அளவாக ஆதி ரகம் டண்ணலோ
ஆழ கோடிகளிடத்தி ற் கி ருவ வைத்து
நளினவகத் தேவருக்கு தேவான
நடேசரே சகலோ ரி டத்தி ற்றாளும்
எளிவாக இயகம வத்தி ற்றானே
இந்தவ ரோசத்தை நீ ர்சொல்லுமென்றாள்

451 (யூகி வைத்தி ய சி ந்தாமணி 800)

It is stated in the above verse that Megha Roga Nidanam was explained to Godess Parvathi by Lord Siva.

வளித்த மேகது இரண்டி டத்து
வாதத்தி ற் பி றந்தசலம் நாலேயாகும்
பி சனித்த பி த்தத்தி லுற்பவித்த
பேரானசலந்தானுமாறு மாகும்
தெசனித்த சேட்டுமத்தி ல் உற்பவித்த
சீ ரானசலந்தானும் டத்தே யாகும்
இசனித்த இனைய குணகுணர்
ஏழி லானஉற் பத்தி யி யம்டக்கேளே

453 (யூகி வைத்தி ய சி ந்தாமணி 800)

Mega diseases are classified into 20 , four comes under Vatha, six under Pitha, ten under Kabam.Under Vatham : Achiya Megham, Suththa Megam, Piramiya Megham, Maangisa ravi megham. Under Pitham : Appiya megham, Aparimiya Megham, Sampoorna Megham, Madhumiya megham, Asathiya megham, Aavirutha megham, Vasa megham, Uthama megham, Macha megham, aakika megham; under kabam : Surari Megham, Sukkila megham, Uthaka megham, Pinani megham, Lavana megham, Thayithiya megham.

சுரரி மேகம்

"பண்ண நுரையோடே வெளுத் தோலப்
பனங்கள்ளுபோலவே தான்றங்கும்
கண்ண வேளைக்கு நாழி யாகும்
காச்சி னல் கள்ளுன்மி கவேவீ சும்
மண்ண மாட்போல மந்தலும்மம்
மயக்கம மிப்படிதான்கண்ட மேகம்
எண்ண ஏழுஎற்றினற் கொல்லும்
ஏற்றமாஞ்சுராரி யென்ற மேந்தானே
(477- யூகி வைத்திய சிந்தாமணி 800)

Voided urine will be frothy and cloudy it may look like toddy, polyuria will be seen. If it is boiled it will smell like toddy. Patient might feel dizzy.These are the characteristics of Surari megham.

வாத மேகம் நாலில் மதுமேக குண

விதமிகுவாத நாலின்மேகீனர் கலீல் தோன்றும்
வதிணையருளா தோன்று மடிக்கடி நீரிறங்கும்
டதியதேனிரத முத்தப் போநீர் நுரைத்திறங்கும்
அிகமாருந்தி ற்றீரா வாத்தியமென்னாமே
(தேரர் குணாகடம் - 37)

Table-6 – Madhumegham breaking of symptoms

மேகீனர் கலீல் தோன்றும்	Heat, pitham
வதிணையருளா தோன்று	Vulvitis, balanitis
மடிக்கடி நீரிறங்கும்	Increased frequency of micturition
டதியதேனிரத முத்தப்	Urine with the taste of fresh honey
நீர் நுரைத்திறங்கும்	Frothy

Patients with diabetes mellitus may be having symptoms of burning micturitiion as a a result of urinary tract infection. Glycosuria can result in vulvitis or balanitis; and there will be

increased frequency of micturitiion. Since there is glycosuria urine will be like honey. And there are chances of froth in urine due to albuminuria.

காசமேகம்

இம்பி ய காச மேகி தேமாய் வாயி னிக்கும்
வியன்பெற முங்கறுக்கும் வெண்ணு நீ ரே வீழும்
அர்ந்துதுங்க முண்ண மரணங் காந்தலாகும்
நயந்தூற்றி ரு ஞண்ண நயந்தவர் நவிற்றுவாரே
(தேரர் குணாகடம் - 30)

Several abnormalities of the respiratory function have been reported in patients with type 1 and type 2 diabetes. These abnormalities concern lung volume, pulmonary diffusing capacity, control of ventilation, bronchomotor tone, and neuroadrenergic bronchial innervation. Many hypotheses have emerged, and characteristic histological changes have been described in the "diabetic lung" (Pitocco et al. 2012) which could explain this abnormal respiratory function.

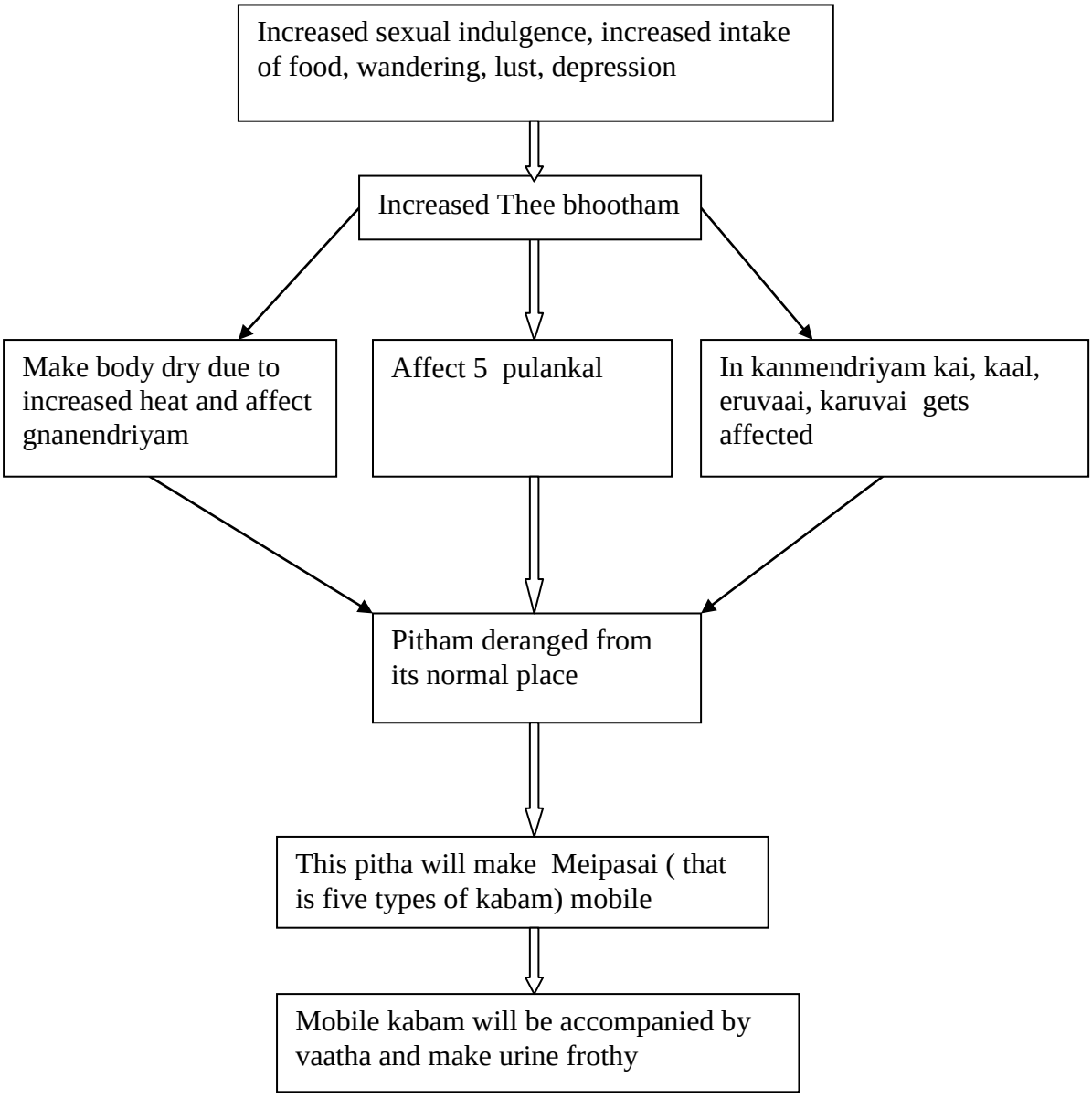
This Kasa megham may be Diabetic Lung condition, due to hyperglycemia there may be sweet taste in mouth. These complications are basically caused by vascular damage, which has a central role in the pathophysiology of diabetes. Despite the presence of a large capillary network in the lung, pulmonary complications of diabetes are frequently disregarded. This is mainly because the alveolar-capillary system is characterized by a great micro vascular reserve, and pulmonary abnormalities are commonly subclinical in diabetic patient. However, the loss of micro vascular reserve in the lung may become clinically important, with increased risk of hypoxia, which may be the reason for blackish discolouration of face. In case of acute or chronic pathological lung conditions, including pneumonia, chronic obstructive pulmonary disease, and asthma, or fluid overload secondary to heart failure . These can show cough as a sign.

சி த்தும தனல் வந்த தீ ரைந்து மி னிநான் சொல்லேன்
சி த்துந் தன்னல் வந்தவந்தாசரப் பி ரமே கந்தான்
சமோற்பம் டச்சி லை வைனாகீ ரைனீர் மீ னிநாற்றமாகும்
மி லாற்பு மற்றி ல் கவுப வலி ற் பி ரமேகமென்றுதுமை சாலுற்றேன்
கழி யீனீர் வெண்ணுயாய் கடத்தங்கே கழி யங் கண்ணாய்
நழி யதாய் ரைந்தி ரதே நாமி துவி ரிந்து சொன்னோம்
இழி யதாய் னீரி ன்சுவை யி னிச்சொல்லேன்நானைப்பி ரமேகம்
வெளியி லே வுைவப் பி ரசரி க்க பி ரசரி க்க வேண்டுங் கண்டுகொள்ளே

(தேரர் குணகடம்)

Piramega noi due to Kabam impairement will show urine with colour like that of a leaf juice, it will smell like fish . Urine would be frothy in nature.

Majoriy of the cases were Diabetic so the root cause of Albuminuria among the subjects has been Diabetes Mellitus(Madhumegham)



The basic constitution of the body is made up of 96 Thathuvams. Due to diet and other activities components of 96 Thathuvams get deranged and result in diseases, either pertaining to body or mind.

In this study majority of the cases with Athi Nurai Neer were diabetic (Madhumegham). As per Siddha literature Mahumegham is caused due to factors such as increased sexual indulgence, increased intake of food, wandering, depression, stress, over ambitious, hereditary factor etc.

DERANGED COMPONENTS OF 96 THATHUVAS ARE AS FOLLOWS

1. AYMBOOTHAMS (FIVE ELEMENTS)

Among Pancha bhootham Theyu bhootham might get deranged initially and because of its derangement it can make the body dry (**உலர்த்தல்**) and it leads to dryness of mouth, burning sensation of soles and palms.

2. IYMPORIGAL (PENTA SENSORS)

Among Iymporigal Kann might get affected vision can get impaired in later stages (diabetic retinopathy). So following porikal pulankal can get affected.

3. KANMENTHIRIYAM/ KANMAVIDAYAM (MOTOR ORGANS)

All five Karmenthiriyangal may get affected, kai, kaal, vai, eruvai, and karuvai might be affected during the progress of the disease. Following diabetic neuropathy both limbs can get affected, dryness of mouth which make vai affected, Eruvai can get affected since there is increased frequency of micturition , like wise diabetics can lead to urinary tract infection and secondary balanoposthitis which makes karuvai affected.

4. NAADI (DIFFERENTIAL PULSE PERCEPTION)

Since "**டீர் பித்த விந்தையாது மேகம் வராதீ**" Pitha may be the main driving cause for Mega diseases. Sangini may be affected because it is located in genito- urinary region.

5. AADHAARAM (STATIONS OF SOUL)

Swathistanam might be affected since genitourinary system is affected

6. PATHINAANGU VEGANGAL (NATURAL URGES/REFLEXES)

Neer (Increased frequency of micturition), Pasi (Increased appetite), Neervetkai (Increased thirst), Ilaippu (Tiredness and weight loss), Sukkilam (Impotency)gets affected

8. AASAYAM:

Salavaasayam (In diabetic nephropathy renal system may get affected), Sukkilavaasayam can also get affected.

9. DERANGED UYIR THATHUKKAL

(HUMORAL OR TRIDOSHA PATHOLOGY)

Panchaboothams manifests in the body as three vital forces, Vatham, Pitham, Kabam

DERANGED OF VATHAM OR VAYU

In Athinurai Neer, primarily affected vayukkal were Abanan, Viyanan, Samaanan Kirukaran and Devathathan.

Table-7 Types of vatham Derangements

Sl No	Affected components of Vatham	Symptoms
1	Abanan	Increased frequency of micturition
2	Viyanan	Movement restriction in diabetic neuropathy
3	Samaanan	Affected since other vaayus are affected
4	Kirukaran	Dryness of mouth
5	Devaduthan	Fatigue

DERANGED OF PITHAM

Pprimarily affected Pitham components can be saathagapitham.

Types of pitham Derangements

Saathagam Difficulty to concentrate work due to weariness of limbs

DERANGED OF KAPHAM

In Athinurai neer primarily affected kabam are Avalambagam and Santhigam

Types of Kabam Derangements

Avalambagam : Santhigam affected hence Avalambakam also gets affected.

Santhigam : Weariness of limbs

10. DERANGED UDAL THATHUKKAL

Saaram	-	Fatigue
Senneer	-	Fatigue
Oon	-	Weariness of limbs
Sukkilam	-	Impotency

11. KOSAM (BODY SYSTEMS)

a) Annamaya kosam – Affected

Anamaya kosam might be affected because 7 Udal thathukkal forming the Kosam are affected.

b) Pranamaya kosam-Affected

Pranamaya kosam can be affected because Kanmenthiriyangal forming this kosam are affected.

c) Manamaya kosam – Affected, since Gnanethiriyangal affected in Diabetic complications.

d) Vignaanamaya kosam – Affected

It is affected because Gnanaenthiriyangal forming this kosam are affected.

KIDNEY

NORMAL STRUCTURE

ANATOMY

The kidneys are bean-shaped paired organs, each weighing about 150 gm in the adult male and about 135 gm in the adult female. The hilum of the kidney is situated at the midpoint on the medial aspect where the artery, vein, lymphatics and ureter are located. The kidney is surrounded by a thin fibrous capsule which is adherent at the hilum. Cut surface of the kidney shows 3 main structures: well demarcated *peripheral cortex, inner medulla* and the innermost *renal pelvis* **the renal cortex** forms the outer rim of the kidney and is about 1 cm in thickness. It contains all the glomeruli and about 85% of the nephron tubules. Remaining 15% nephrons consisting of collecting tubules, collecting ducts, loops of Henle and vasa recta send their loops into the medulla, and are therefore called juxtamedullary nephrons. This latter part of the cortex forms faint striations called *medullary rays,* a misnomer since theses structures are located in the cortex but are destined for medulla. Columns of renal cortical tissue that extend into the space between adjacent pyramids are called the *renal column (septa) of Bertin;* they contain the interlobar arteries. The **renal medulla** is composed of 8-18 cone-shaped renal pyramids. The base of a renal pyramid lies adjacent to the outer cortex and forms the cortico-medullary junction, while the apex of each called the *renal papilla* contains the opening of each renal pyramid for passage of urine collected from collecting ducts and goes down into minor calyces. The **renal pelvis** is the funnel-shaped collection area of the urine for drainage into the ureter. The minor calyces (8- 18 in number in a normal kidney) collect urine from renal papillae and drain into major calyces (2-3 in a normal kidney).

HISTOLOGY.

The parenchyma of each kidney is composed of approximately one million microstructures called nephrons. A nephron, in turn, consists of 5 major parts, each having a functional role in the formation of urine: the glomerular capsule (glomerulus and Bowman's capsule), the proximal convoluted tubule (PCT), the loop of Henle, the distal convoluted tubule (DCT), and the collecting ducts. From point of view of diseases of the kidneys, 4 components of renal parenchyma require further elaboration: renal vasculature, glomeruli, tubules and interstitium.

1. Renal vasculature. Each kidney is supplied with blood by a main *renal artery* which arises from the aorta at the level of the 2nd lumbar vertebra. It usually divides into *anterior and posterior divisions* at the hilum although occasionally these divisions may even arise directly from the aorta. The anterior and posterior divisions divide into *segmental branches* from which *interlobar arteries* arise which course between the lobes. Along their course, they give off the *arcuate arteries* which arch between the cortex and medulla. The arcuate arteries, in turn, give off *interlobular arteries* which lie in the cortex perpendicular to the capsular surface in the part overlying the pyramids and, therefore, are also called *straight arteries*

Systemic Pathology

Arterioles take their origin, each one supplying a single glomerulus. From the glomerulus emerge the *efferent arterioles*. Up to this stage, the arteries and arterioles are endvessels. The efferent arterioles leaving the glomerulus supply *peritubular capillary plexus* which anastomoses with the capillary plexus of another nephron. The juxtamedullary glomeruli, however, give off a series of parallel vessels called *vasa recta* which descend to the inner medulla supplying the loop of Henle and collecting ducts and anastomose at all levels throughout the medulla with the ascending vasa recta. These drain into *arcuate veins* and then into the veins that accompany the corresponding arteries and finally through a single renal vein into the inferior vena cava . Lymphatic drainage likewise occurs through lymphatics associated with the intrarenal vasculature leaving the kidney at the hilum and draining to lateral aortic lymph nodes. The following important inferences can be drawn from the peculiarities of the renal vasculature: i) The renal cortex receives about 90% of the total renal blood supply and that the pressure in the glomerular capillaries is high. Therefore, renal cortex is more prone to the effects of hypertension. ii) The renal medulla, on the other hand, is poorly perfused and any interference in blood supply to it results in medullary necrosis. iii) The divisions and subdivisions of the renal artery up to arterioles are end-arteries and have no anastomoses. Thus, occlusion of any of the branches results in infarction of the renal parenchyma supplied by it. iv) Since the tubular capillary beds are derived from the efferent arterioles leaving the glomeruli, diseases affecting the blood flow through glomerular tuft have significant effects on the tubules as well.

2. Glomerulus. The glomerulus consists of invagination of the blind end of the proximal tubule and contains a *capillary tuft* fed by the afferent arteriole and drained by efferent arteriole. The capillary tuft is covered by visceral epithelial cells (podocytes) which are continuous with those of the parietal epithelium at the *vascular pole.* The transition to proximal tubular cells occurs at the *urinary pole* of the glomerulus. The visceral and parietal epithelial cells are separated by the urinary space or *Bowman's space,* into which glomerular filtrate passes **.**Subdivisions of capillaries derived from the afferent arterioles result in the formation of *lobules* (up to 8 in number) within a glomerulus. Each lobule of a glomerular tuft consists of a centrilobular supporting stalk composed of mesangium containing *mesangial cells* and *mesangial matrix.* The mesangium is continuous at the hilum with the *lacis cells* of the juxtaglomerular apparatus. Besides their role as supportive cells, mesangial cells are involved in the production of mesangial matrix and glomerular basement membrane; they function in endocytosis of leaked macromolecules and also possibly in the control of glomerular blood flow through contractile elements present in these cells. The major *function* of glomerulus is complex filtration from the capillaries to the urinary space. Glomerular filtrate is quite similar in composition to plasma but lacks proteins and cells. Normally, glomerular filtration rate (GFR) is about 125 ml/minute. The barrier to glomerular filtration consists of the following 3 components i) Fenestrated endothelial cells lining the capillary loops. ii) Glomerular basement membrane (GBM) on which the endothelial cells rest. It further consists of 3 layers—the central lamina densa, bounded by lamina rara interna on endothelial side of the capillary and lamina rara externa on visceral epithelial side of the capillary. iii) Filtration slit pores between the foot processes of the visceral epithelial cells (podocytes) external to GBM. The barrier to filtration of macromolecules of the size and molecular weight of albumin and larger depends upon the following: A normal lamina densa. Maintenance of negative charge on both lamina rarae.A healthy covering of glomerular epithelial cells.**Juxtaglomerular apparatus.**The juxtaglomerular apparatus (JGA) is situated at the vascular pole of the glomerulus and is made up of 3 parts,.

2 The Kidney and Lower Urinary Tract i) The*juxtaglomerular cells* are modified granular smooth muscle cells in the media of the afferent arteriole and contain the hormone, renin. ii) The *macula densa* is comprised by specialised region of the distal tubule when it returns to the vascular pole of its parent glomerulus. The tubular cells here are taller and narrower than elsewhere with the nuclei lying close together. iii) The *lacis cells* or *non-granular cells* occupy the space between the macula densa and the arterioles and merge with the glomerular

mesangium. The JGA is intimately concerned with sodium metabolism and is the principal source of renin production. The mechanism of the release of renin and its role in hypertension.

3. Tubules. The tubules of the kidney account for the greatest amount of the renal parenchyma. The structure of renal tubular epithelium varies in different parts of the nephron and is correlated with the functional capacity of that part of the tubule

i) Proximal convoluted tubule (PCT). This is the first part arising from the glomerulus and is highly specialised part functionally. It is lined by cuboidal cells with a brush border composed of microvilli and contains numerous mitochondria, Golgi apparatus and endoplasmic reticulum. The major functions of PCT are: *active reabsorption* of filtered sodium, potassium, glucose, amino acids, proteins, vitamins, bicarbonate, phosphate, calcium and uric acid, and *passive reabsorption* of 80% of filtered water.

ii) Loop of Henle. The PCT drains into the straight part of loop of Henle that consists of thin descending, and thin and thick ascending limbs, both of which have different structure and function. The descending limb is continuation of PCT, while ascending limb continues further into distal convoluted tubule (DCT). The descending segment of loop is lined by simple epithelium while the ascending limb is lined by columnar cells. The major function of loop of Henle is *active reabsorption* of sodium, potassium and chloride, and *passive diffusion* of water resulting in concentrated filtrate of urine.

iii) Distal convoluted tubule (DCT). The DCT represents a transition from thick ascending limb from the point where the ascending limb meets the vascular pole of the glomerulus of its origin, to the early collecting ducts.

Systemic Pathology

DCT are cuboidal. The epithelial cells at the point of beginning of DCT are taller, narrower and more closely packed to form the macula densa of JGA as already described. The DCT further contributes to urinary concentration and acidification, while the macula densa of JGA is the source of renin and has a role in sodium metabolism. *iv) Collecting ducts.* The system of collecting ducts is the final pathway by which urine reaches the tip of renal papilla. The cells lining the collecting ducts are cuboidal but lack the brush border. Collecting ducts reabsorb water under control of ADH, and secrete H+ and K+ ions.

4. Interstitium. In health, the renal cortical interstitium is scanty and consists of a small number of fibroblast-like cells. But the medullary interstitium is more plentiful and contains stellate interstitial cells which are considered to produce an anti-hypertensive agent and are involved in the metabolism of prostaglandins.

RENAL FUNCTION TESTS

In general, the kidney performs the following vital functions in the body:

1. *Excretion of waste products* resulting from protein metabolism.
2. *Regulation of acid-base balance* by excretion of H+ ions (acidification) and bicarbonate ions.
3. *Regulation of salt-water balance* by hormones secreted both intra- and extra-renally.
4. *Formation of renin and erythropoietin* and thereby playing a role in the regulation of blood pressure and erythropoiesis respectively. In order to assess renal function, a number of tests have been devised which give information regarding the following parameters:

a) Renal blood flow

b) Glomerular filtration

c) Renal tubular function

d) Urinary outflow unhindered by any obstruction.

Renal function tests are broadly divided into 4 groups

1. Urine analysis.
2. Concentration and dilution tests.
3. Blood chemistry.
4. Renal clearance tests.

In addition, renal biopsy is performed to confirm the diagnosis of renal disease. Renal biopsy is ideally fixed in alcoholic Bouin's solution and examined by routine morphology combined with special stains and further studies as under:

1. *Periodic acid-Schiff* stain for highlighting glomerular basement membrane.
2. *Silver impregnation* to outline the glomerular and tubular basement membrane.
3. *Immunofluorescence* to localise the antigens, complements and immunoglobulins.
4. *Electron microscopy* to see the ultrastructure of glomerular changes.

1. URINE ANALYSIS. The simplest diagnostic tests for renal function are the physical, chemical, bacteriologic and microscopic examination of the urine. i) The *physical examination* includes 24-hour urinary output, colour, specific gravity and osmolality. Normally urine is clear,

pale or straw-coloured due to pigment urochrome and 700-2500 ml (average 1200 ml) of urine is passed in 24 hours, mostly during day time. Specific gravity is used to measure the concentrating and diluting power of the kidneys.

ii) The *chemical tests* are carried out to detect the presence of protein, glucose, red cells and hemoglobin to assess the permeability of glomerular membrane. A number of convenient dipstick tests are available for testing these chemical substances and pH. These consist of paper strips impregnated with appropriate reagents and indicator dyes.

ii) The *bacteriologic examination* of the urine is done by proper and aseptic collection of midstream specimen of urine.

iv) *Urine microscopy* is undertaken on a fresh unstained sample. Various components observed on microscopic examination of the urine in renal disease are red cells, pus cells, epithelial cells, crystals and urinary casts. The casts are moulded into cylindrical shapes by passage along tubules in which they are formed. They are the result of precipitation of proteins in the tubule that includes not only albumin but also the tubular secretion of the *Tamm Horsfall protein*. The latter is a high molecular weight glycoprotein normally secreted by ascending loop of Henle and DCT and probably has body defense function normally. Its secretion is increased in glomerular and tubular diseases. Casts may be *hyaline type* consisting of only proteins indicating a non-inflammatory etiology of glomerular filtration of proteins, *leucocyte casts* inflammatory in origin, or *red cell casts* from haematuria.

2. CONCENTRATION AND DILUTION TESTS.

Concentration and dilution tests are designed to evaluate functional capacity of the renal tubules. The ability of the nephron to concentrate or dilute urine is dependent upon both functional activity of the tubular cells in the renal medulla and the

Renal Function Tests.

1. URINE ANALYSIS:

i) Physical examination (output, colour, specific gravity, pH, osmolality)

ii) Chemical constituents (protein, glucose, red cells, haemoglobin)

iii) Bacteriologic examination

iv) Microscopy

2. CONCENTRATION AND DILUTION TESTS:

i) Concentration test (fluid deprivation test)

ii) Dilution test (excess fluid intake test)

3. BLOOD CHEMISTRY:

i) Urea

ii) Blood urea nitrogen (BUN)

iii) Creatinine

4. RENAL CLEARANCE TEST:

i) Inulin or mannitol clearance test

ii) Creatinine clearance

iii) Urea clearance

iv) Para-aminohippuric acid (PAH) clearance

Failure to achieve adequate urinary concentration can be due to either defects within the renal medulla *(nephrogenic diabetes insipidus),* or due to the lack of ADH *(central diabetes insipidus).* Traditionally, urinary concentration is determined by specific gravity of the urine (normal range 1.003 to 1.030, average 1.018) which in cases of tubular disease remains constant at approximately 1.010 regardless of changing levels of plasma hydration. However, determination of urinary specific gravity provides only a rough estimate of osmolarity of the urine. The tubular disease can be diagnosed in its early stage by *water deprivation* (concentration) or *water excess* (dilution) tests.

i) In **concentration test,** an artificial fluid deprivation is induced in the patient for more than 20 hours. If the nephron is normal, water is selectively reabsorbed resulting in excretion of urine of high solute concentration (specific gravity of 1.025 or more). However, if the tubular cells are

nonfunctional, the solute concentration of the urine will remain constant regardless of stress of water deprivation.

ii) In **dilution test,** an excess of fluid is given to the patient. Normally, renal compensation should result in excretion of urine with high water content and lower solute concentration (specific gravity of 1.003 or less). If the renal tubules are diseased, the concentration of solutes in the urine will remain constant irrespective of the excess water intake.

3. BLOOD CHEMISTRY. Impairment of renal function results in elevation of end-products of protein metabolism. This includes increased accumulation of certain substances in the blood, chiefly urea (normal range 20-40 mg/dl), blood urea nitrogen (BUN) (normal range 10-20 mg/dl) and creatinine (normal range 0.6-1.2 mg/dl). An increase of these end-products in the blood is called *azotaemia.* High levels of creatinine are associated with high levels of 2-*microglobulin* in the serum as well as urine, a lowmolecular weight protein filtered excessively in the urine due to glomerular disease or due to increased production by the liver.

4. RENAL CLEARANCE TESTS. A clearance test is employed to assess the rate of glomerular filtration and the renal blood flow. The rate of this filtration can be measured by determining the excretion rate of a substance which is filtered through the glomerulus but subsequently is neither reabsorbed nor secreted by the tubules. The glomerular filtration rate (normal 120 ml/minute in an average adult) is usually equal to clearance of that substance and is calculated from the following equation:

$$C = \frac{UV}{P} \; where$$

C is the clearance of the substance in ml/ minute;
U is the concentration of the substance in the urine;
V is the volume of urine passed per minute; and
P is the concentration of the substance in the plasma.

The substances which are used for clearance tests include inulin, mannitol, creatinine and urea.

i) In **inulin or mannitol clearance tests,** an intravenous infusion of the substance inulin or mannitol is given to maintain constant plasma concentration and accurately timed urine samples

are collected. Inulin, a mixture of fructose polymers, is considered the ideal substance for the clearance test since it is filtered from the glomerulus and is excreted unchanged in the urine.

ii) In **creatinine clearance test,** there is no need of intravenous infusion of creatinine since creatinine is normally released into plasma by muscle metabolism and a very small fraction of this substance is secreted by the tubules. The clearance of creatinine is determined by collecting urine over 24-hour period and a blood sample is withdrawn during the day. In spite of disadvantages like poor reproducibility and secretion of creatinine by the tubules, the 'endogenous' creatinine clearance test is easy and routinely employed method of estimating GFR.

iii) In **urea clearance test,** the sensitivity is much less than the creatinine or inulin clearance because plasma concentration of urea is affected by a number of factors (e.g. dietary protein, fluid intake, infection, trauma, surgery, and corticosteroids) and is partly reabsorbed by the tubules. Like in creatinine clearance, there is no need for intravenous infusion of urea.

iv) **Para-aminohippuric acid (PAH) clearance test** is employed to measure renal blood flow (unlike the preceding tests which measure GFR). PAH when infused intravenously is both filtered at the glomerulus as well as secreted by the tubules and its clearance is measured by determining its concentration in arterial blood and urine. Normally, renal blood flow is about 1200 ml per minute in an average adult.

PATHOPHYSIOLOGY OF RENAL DISEASE: **RENAL FAILURE**

Traditionally, diseases of the kidneys are divided into 4 major groups according to the predominant involvement of corresponding morphologic components:

1. *Glomerular diseases:* These are most often immunologically- mediated and may be acute or chronic.

2. *Tubular diseases:* These are more likely to be caused by toxic or infectious agents and are often acute.

3. *Interstitial diseases:* These are likewise commonly due to toxic or infectious agents and quite often involve interstitium as well as tubules (tubulo-interstitial diseases).

4. *Vascular diseases:* These include changes in the nephron as a consequence of increased intra-glomerular pressure such as in hypertension or impaired blood flow. include: congenital anomalies, obstructive uropathy (including urolithiasis) and tumours of the kidneys. The major morphologic involvements of the kidneys in the initial stage is confined to one component (glomeruli, tubules, interstitium or blood vessels), but eventually all components are affected leading to *end-stage kidneys.* Regardless of cause, renal disease usually results in the evolution of one of the two major pathological syndromes: *acute renal failure* and *chronic renal failure.*

Proteinuria is the presence of excess proteins in the urine. In healthy persons, urine contains very little protein; an excess is suggestive of illness. Excess protein in the urine often causes the urine to become foamy.

There are three main mechanisms to cause proteinuria:

- Due to disease in the glomerulus
- Because of increased quantity of proteins in serum (overflow proteinuria)
- Due to low reabsorption at proximal tubule

Proteinuria may be a sign of renal (kidney) damage. Since serum proteins are readily reabsorbed from urine, the presence of excess protein indicates either an insufficiency of absorption or impaired filtration. People with diabetes may have damaged nephronsand develop proteinuria. The most common cause of proteinuria is diabetes, and in any person with proteinuria and diabetes, the cause of the underlying proteinuria should be separated into two categories: diabetic proteinuria versus the field.

Conditions with proteinuria as a sign

Proteinuria may be a feature of the following conditions:

- Nephrotic syndromes (i.e. intrinsic renal failure)
- Pre-eclampsia
- Eclampsia
- Toxic lesions of kidneys
- Amyloidosis
- Collagen vascular diseases (e.g. systemic lupus erythematosus)
- Dehydration
- Glomerular diseases, such as membranous glomerulonephritis, focal segmental glomerulonephritis, minimal change disease (lipoid nephrosis)
- Strenuous exercise

- Stress
- Benign orthostatic (postural) proteinuria
- Focal segmental glomerulosclerosis (FSGS)
- IgA nephropathy (i.e. Berger's disease)
- IgM nephropathy
- Membranoproliferative glomerulonephritis
- Membranous nephropathy
- Minimal change disease
- Sarcoidosis
- Alport's syndrome
- Diabetes mellitus (diabetic nephropathy)
- Drugs (e.g. NSAIDs, nicotine, penicillamine, lithium carbonate, gold and other heavy metals, ACE inhibitors, antibiotics, or opiates (especially heroin)[7]
- Fabry's disease
- Infections (e.g. HIV, syphilis, hepatitis, poststreptococcal infection, urinary schistosomiasis)
- Aminoaciduria
- Fanconi syndrome in association with Wilson disease
- Hypertensive nephrosclerosis
- Interstitial nephritis
- Sickle cell disease
- Hemoglobinuria
- Multiple myeloma
- Myoglobinuria
- Nail patella syndrome
- Systemic lupus erythematosus
- Goodpasture's syndrome
- Henoch–Schönlein purpuras
- A urinary tract infection which has spread to the kidney(s)
- Post-infectious glomerulonephritis

Nephrotic Syndromes

Nephrotic syndrome is a collection of symptoms due to kidney damage and is primarily a paediatric disorder and is 15 times more common in children than in adults. The incidence is 2-3/100,000 children per year; and the majority of affected children will have steroid sensitive minimal change disease. The characteristic features of nephritic syndrome are heavy protienuria

>3.5g/24 hr in adults or 40 mg/m^2/hr in children, hypoalbuminemia, oedema, and hyperlipidemia. (1)

Causes of Nephrotic Syndrome.

I. PRIMARY GLOMERULONEPHRITIS

1. Minimal change disease *(most common in children)*
2. Membranous GN *(most common in adults)*
3. Membranoproliferative GN
4. Focal segmental glomerulosclerosis
5. Focal GN
6. IgA nephropathy

II. SYSTEMIC DISEASES

1. Diabetes mellitus
2. Amyloidosis
3. SLE

III. SYSTEMIC INFECTIONS

1. Viral infections (HBV, HCV, HIV)
2. Bacterial infections (bacterial endocarditis, syphilis, leprosy)
3. Protozoa and parasites (*P. falciparum* malaria, filariasis)

IV. HYPERSENSITIVITY REACTIONS

1. Drugs (heavy metal compounds like gold and mercury, other drugs like penicillamine, trimethadione and tolbutamide, heroin addiction)

2. Bee stings, snake bite, poison ivy

V. MALIGNANCY

1. Carcinomas
2. Myeloma
3. Hodgkin's disease

VI. PREGNANCY

Toxaemia of pregnancy

VII. CIRCULATORY DISTURBANCES

1. Renal vein thrombosis
2. Constrictive pericarditis

VIII. HEREDITARY DISEASES

1. Alport's disease
2. Fabry's disease
3. Nail-patella syndrome

NEPHROTIC SYNDROME Nephrotic syndrome is a constellation of features in different diseases having varying pathogenesis; it is characterised by findings of massive proteinuria, hypoalbuminaemia, oedema, hyperlipidaemia, lipiduria, and hypercoagulability.

1. Heavy proteinuria (protein loss of more than 3 gm per 24 hrs) is the chief characteristic of nephrotic syndrome *(nephrotic range proteinuria).* In children, protein loss is correspondingly less. A small amount of protein (20 to 150 mg/day) normally passes through the glomerular filtration barrier and is reabsorbed by the tubules. But in case of increased glomerular permeability to plasma proteins, excess of protein is filtered out exceeding the capacity of tubules for reabsorption and, therefore, appears in the urine. Another feature of protein loss is its 'selectivity'. A *highly-selective proteinuria* consists mostly of loss of low molecular weight proteins, while a *poorly-selective proteinuria* is loss of high molecular weight proteins in the urine. In nephritic syndrome, proteinuria mostly consists of loss of albumin (molecular weight 66,000) in the urine.

2. Hypoalbuminaemia is produced primarily consequent to urinary loss of albumin, and partly due to increased renal catabolism and inadequate hepatic synthesis of albumin Often, the plasma albumin level is 1 to 3 gm/dl (normal 3.5 to 5.5 gm/dl) and there is reversed albumin-globulin ratio.The concentration of other proteins in the plasma such as immunoglobulins, clotting factors and antithrombin may fall rendering these patients more vulnerable to infections and thrombotic and thromboembolic complications.

3. Oedema in nephrotic syndrome appears due to fall in colloid osmotic pressure consequent upon hypoalbuminaemia. Sodium and water retention further contribute to oedema. Nephrotic oedema is usually peripheral but in children facial oedema may be more prominent .

4. Hyperlipidaemia is a frequent accompaniment of nephrotic syndrome. The exact mechanism of its genesis is not clear. It is hypothesised that the liver faced with the stress of massive protein synthesis in response to heavy urinary protein loss, also causes increased synthesis of lipoproteins. There are increased blood levels of total lipids, cholesterol, triglycerides, VLDL and LDL but decrease in HDL. Low blood level of HDL is partly due to its loss in the urine.

5. Lipiduria occurs following hyperlipidaemia due to excessive leakiness of glomerular filtration barrier.

6. Hypercoagulability. Patients with nephrotic syndrome may develop spontaneous arterial or venous thrombosis, renal vein thrombosis and pulmonary embolism due to various factors. These include: increased urinary loss of antithrombin III, hyperfibrinogenaemia from increased synthesis in the liver, decreased fibrinolysis, increased platelet aggregation and altered levels of protein C and S. The causes of nephrotic syndrome are diverse .The morphology of individual types is described later. But it must be mentioned here that: in *children,* primary glomerulonephritis is the cause in majority of cases of the nephrotic syndrome; most frequent is *lipoid nephrosis (65%);* and *in adults,* on the other hand, systemic diseases (diabetes, amyloidosis and SLE) are more frequent causes of nephritic syndrome. The most common primary glomerular disease in adults is *membranous glomerulonephritis* (40%).

III.ACUTE RENAL FAILURE. As already described above, acute renal failure (ARF) is characterised by rapid decline in renal function. ARF has many causes including glomerular disease, principally rapidly progressive GN and acute diffuse proliferative GN.

IV.CHRONIC RENAL FAILURE. Glomerular causes of chronic renal failure (CRF) have already been described. These cases have advanced renal impairment progressing over years and is detected by significant proteinuria, haematuria, hypertension and azotaemia. Such patients generally have small contracted kidneys due to chronic glomerulonephritis.

The renal glomerulus filters the blood that arrives at the kidney. It is formed of capillaries with small pores that allow small molecules to pass through that have a molecular weight of less than 40,000 Daltons, but not larger macromolecules such as proteins.

In nephrotic syndrome, the glomeruli are affected by an inflammation or a *hyalinization* (the formation of a homogenous crystalline material within cells) that allows proteins such

as albumin, antithrombin or the immunoglobulins to pass through the cell membrane and appear in urine.

Albumin is the main protein in the blood that is able to maintain an oncotic pressure, which prevents the leakage of fluid into the extracellular medium and the subsequent formation of edemas.

As a response to hypoproteinemia the liver commences a compensatory mechanism involving the synthesis of proteins, such as alpha-2 macroglobulin and lipoproteins. An increase in the latter can cause the hyperlipidemia associated with this syndrome.

Etiology

Most children with nephritic syndrome have a form of the idiopathic nephritic syndrome.Causes of idiopathic nephritic syndrome include minimal change disease (85%), mesangial proliferation (5%) and focal segmental glomerulosclerosis (10%). The remaining 10% of children with nephrotic syndrome have secondary nephritic syndrome related to systemic or glomerular diseases such as membranous nephropathy or membranoproliferative glomerulonephritis.

Nephrotic syndrome has many causes and may either be the result of a glomerular disease that can be either limited to the kidney, called *primary* nephrotic syndrome (primary glomerulonephrosis), or a condition that affects the kidney and other parts of the body, called *secondary*nephrotic syndrome.

AMYLOIDOSIS

Amyloidosis of kidneys is most common and most serious because of ill effects on renal function. The deposits in the kidneys are found in most cases of secondary amyloidosis and in about one-third cases of primary amyloidosis. Amyloidosis of kidney accounts about 20% deaths from amyloidosis. Even small quantities of amyloid deposits in the glomeruli can cause protienuria and nephritic syndrome..In the glomeruli, the deposits initially appear on the basement membrane of the glomerular capillaries, but later extend to produce luminal narrowing and distortion of the glomerular capillary tuft. This results in abnormal increase in permeability of the glomerular capillaries to macromolecules with consequent protienuria and nephritic syndrome.

PRE-ECLAMPSIA AND ECLAMPSIA

The exact nature of pathogenesis of pre-eclampsia remains uncertain. Nearly every major system in the body is affected by the advanced manifestations of the condition and therefore every system that is studied appears to show changes without necessarily doing more than manifesting secondary effects.

There is a sequence of events leading to the development of pre-eclampsia , abnormal implantation leads to underperfusion of the placenta which produces an agent that damages the endothelium. The damaged endothelium loses its anticoagulant properties and activates the coagulation cascade. The damaged endothelium is also more sensitive to circulating pressors producing vasospasm, hypertension, and reduced end-organ perfusion. The damaged endothelium is also more permeable, resulting in a shift of intravascular fluid to the interstitium-oedema – and leak of protein through the glomerulus – protienuria.

COLLAGEN VASCULAR DISEASES

Renal involvement in systemic lupus erythematosus (SLE), dysproteinemias, and certain rheumatic diseases, namely rheumatoid arthritis, Sjögren's syndrome, and scleroderma (systemic sclerosis), is discussed. SLE is a systemic autoimmune disease that can lead to disease manifestations in almost every organ. SLE is characterized by the formation of a wide array of autoantibodies mainly directed against nuclear autoantigens, of which antibodies against double-stranded DNA (dsDNA) are the most prominent. Although the cause is still obscure, considerable progress has been made recently by identification of the nucleosome as the major driving autoantigen in SLE and the possible role of disturbances in apoptosis in disease development.

Incidence of renal manifestations and serologic abnormalities in the different forms of lupus nephritis. The clinical manifestations of lupus nephritis are not different from other forms of glomerulonephritis and include a nephritic sediment (dysmorphic erythrocytes and erythrocyte casts), proteinuria or nephrotic syndrome, impaired renal function, and hypertension.

GOODPASTURE'S SYNDROME

Goodpasture's syndrome or pulmonary haemorrhage syndrome is combination of necrotising haemorrhagic interstitial pneumonitis and rapidly progressive glomerulonephritis.

Etiopathogenesis. The condition results from immunologic damage produced by anti-basement membrane antibodies formed against antigens common to the glomerular and pulmonary basement membranes. The trigger for initiation of this autoimmune response is not clear; it could be virus infection, exposure to hydrocarbons and smoking.

Morphologic features. Grossly, the lungs are heavy with red-brown areas of consolidation.

Microscopically, the features vary according to the stage of the disease: *In acute stage,* there are focal areas of haemorrhages in the alveoli and focal necrosis in the alveolar walls. *In more chronic cases,* there is organisation of the haemorrhage leading to interstitial fibrosis and filling of alveoli with haemosiderin-laden macrophages.

Clinical features. The condition occurs commonly in 2nd or 3rd decades of life with preponderance in males. The pulmonary manifestations generally precede the renal disease. Most cases present with haemoptysis accompanied with dyspnoea, fatigue, weakness and anaemia. Renal manifestations soon appear which include haematuria, proteinuria, uraemia and progressive renal failure

LUPUS NEPHRITIS

Renal manifestations of systemic lupus erythematosus (SLE) are termed lupus nephritis. Other clinical manifestations, etiology and pathogenesis of this multi-system autoimmune disease are described in Chapter 4 (page 78). The incidence of renal involvement in SLE ranges from 40 to 75%. The two cardinal clinical manifestations of lupus nephritis are proteinuria and haematuria. In addition, hypertension and casts of different types such as red cell casts, fatty casts and leucocyte casts in the urinary sediment are found. Pathogenesis of lesions in lupus nephritis is linked to genes related to major histocompatibility complex and B-cell signaling pathways such as TNF superfamily members

TUBERCULOUS PYELONEPHRITIS

Tuberculosis of the kidney occurs due to haematogenous spread of infection from another site, most often from the lungs. Less commonly, it may result from ascending infection from tuberculosis of the genitourinary system such as from epididymis or Fallopian tubes. The renal lesions in tuberculosis may be in the form of tuberculous pyelonephritis or appear as multiple miliary tubercles.

Morphologic features.

Grossly, the lesions in tuberculous pyelonephritis are often bilateral, usually involving the medulla with replacement of the papillae by caseous tissue. Obstruction may result in tuberculous pyonephrosis in which thinned out renal parenchyma surrounds dilated pelvis and calyces filled with caseous material.

Histologically, typical granulomatous reaction is seen. Acid-fast bacilli can often be demonstrated in the lesions.

Clinical features. Most patients are young to middleaged adults. The clinical presentation is extremely variable but it should always be considered as a possibility in a patient in whom there is persistent sterile pyouria, microscopic haematuria and mild proteinuria after effective antibiotic therapy for urinary tract infection. The diagnosis rests on identification of *M. tuberculosis* by repeated culture of urine on L.J. media.

ASYMPTOMATIC PROTEINURIA. Presence of proteinuria unexpectedly in a patient may be unrelated to renal disease (e.g. exercise-induced, extreme lordosis and orthostatic proteinuria), or may indicate an underlying mild glomerulonephritis. Association of asymptomatic haematuria, hypertension or impaired renal function with asymptomatic proteinuria should raise strong suspicion of underlying glomerulonephritis.

DIABETIC NEPHROPATHY

Renal involvement is an important complication of diabetes mellitus. End-stage kidney with renal failure accounts for deaths in more than 10% of all diabetics. Renal complications are more severe, develop early and more frequently in type 1 (earlier called insulin-dependent) diabetes mellitus (30-40% cases) than in type 2 (earlier termed non-insulindependent) diabetics (about 20% cases). A variety of clinical syndromes are associated with diabetic nephropathy that includes asymptomatic proteinuria, nephrotic syndrome, progressive renal failure and hypertension. Cardiovascular disease is 40 times more common in patients of end-stage renal disease in diabetes mellitus than in non-diabetics and more diabetics die from cardiovascular complications than from uraemia.

Morphologic features. Diabetic nephropathy encompasses 4 types of renal lesions in diabetes mellitus: diabetic glomerulosclerosis, vascular lesions, diabetic pyelonephritis and tubular lesions (Armanni-Ebstein lesions).

1. DIABETIC GLOMERULOSCLEROSIS. Glomerular lesions in diabetes mellitus are particularly common and account for majority of abnormal findings referable to the kidney. *Pathogenesis* of these lesions in diabetes mellitus is explained by following sequential changes:

- hyperglycaemia
- glomerular hypertension
- renal hyperperfusion
- deposition of proteins in the mesangium
- glomerulosclerosis
- renal failure.

In addition, cellular infiltration in renal lesions in diabetic glomerular lesions is due to growth factors, particularly transforming growth factor. Strict control of blood glucose level and control of systemic hypertension in these patients retards progression to diabetic nephropathy. Glomerulosclerosis in diabetes may take one of the 2 forms: diffuse or nodular lesions:

i) Diffuse glomerulosclerosis. Diffuse glomerular lesions are the most common. There is involvement of all parts of glomeruli. The pathologic changes consist of thickening of the GBM and diffuse increase in mesangial matrix with mild proliferation of mesangial cells. Various exudative lesions such as capsular hyaline drops and fibrin caps may also be present *Capsular drop* is an eosinophilic hyaline thickening of the parietal layer of Bowman's capsule and bulges into the glomerular space. *Fibrin cap* is homogeneous, brightly eosinophilic material appearing on the wall of a peripheral capillary of a lobule.

ii) Nodular glomerulosclerosis. Nodular lesions of diabetic glomerulosclerosis are also called as *Kimmelstiel- Wilson (KW) lesions* or *intercapillary glomerulosclerosis.*These lesions are specific for type 1 diabetes (juvenileonset diabetes) or islet cell antibody-positive diabetes mellitus. The pathologic changes consist of one or more nodules in a few or many glomeruli. *Nodule* is an ovoid or spherical, laminated, hyaline, acellular mass located within a lobule of the glomerulus. The nodules are surrounded peripherally by glomerular capillary loops which may have normal or thickened GBM The nodules are PAS-positive and contain lipid and fibrin. As

the nodular lesions enlarge, they compress the glomerular capillaries and obliterate the glomerular tuft **.**As a result of glomerular and arteriolarinvolvement, renal ischaemia occurs leading to tubular atrophy and interstitial fibrosis and grossly small, contracted kidney.

VASCULAR LESIONS.

Atheroma of renal arteries is very common and severe in diabetes mellitus. ***Hyaline arteriolosclerosis*** affecting the afferent and efferent arterioles of the glomeruli is also often severe in diabetes. These vascular lesions are responsible for renal ischaemia that results in tubular atrophy and interstitial fibrosis.

DIABETIC PYELONEPHRITIS

Poorly-controlled diabetics are particularly susceptible to bacterial infections. Papillary necrosis (necrotising papillitis) is an important complication of diabetes that may result in acute pyelonephritis. Chronic pyelonephritis is 10 to 20 times more common in diabetics than in others.

HEREDITARY NEPHRITIS

A group of hereditary diseases principally involving the glomeruli are termed hereditary nephritis. These include the following:

1. Alport's syndrome
2. Fabry's disease
3. Nail-patella syndrome

1. Alport's syndrome. Out of various hereditary nephritis, Alport's syndrome is relatively more common and has been extensively studied. This is an X-linked dominant disorder having mutation in □ −5 chain of type IV collagen located on X-chromosome. It affects males more severely than females. The syndrome consists of sensori-neural deafness and ophthalmic complications (lens dislocation, posterior cataracts and corneal dystrophy) associated with hereditary nephritis. The condition is slowly progressive, terminating in end-stage kidney in the 2nd to 3rd decades of life. The common presenting features are persistent or recurrent haematuria accompanied by erythrocyte casts, proteinuria and hypertension.

Fabry's disease, another hereditary nephritis is characterised by accumulation of neutral glycosphingolipids in lysosomes of glomerular, tubular, vascular and interstitial cells.

Nail-patella syndrome or osteonychodysplasia is a rare hereditary disease having abnormality in 1 chain of collagen V on chromosome 9 associated with multiple
osseous defects of elbows, knees and nail dysplasia. About half the cases develop nephropathy.

MINIMAL CHANGE DISEASE *(Synonyms:* MCD*, Lipoid Nephrosis, Foot Process Disease, Nil Deposit Disease)*

Minimal change disease (MCD) is a condition in which the nephrotic syndrome is accompanied by no apparent change in glomeruli by light microscopy. Its other synonyms, lipoid nephrosis and foot process disease, are descriptive terms for fatty changes in the tubules and electron microscopic appearance of flattened podocytes respectively. Minimal change disease accounts for 80% cases of nephrotic syndrome
in children under 16 years of age with preponderance in boys (ratio of boys to girls 2:1). In fact, historically, lipoid nephrosis was the first condition associated with nephrotic sndrome.

Etiopathogenesis.

The etiology of MCD remain elusive. However, following two groups have been identified:
i) Idiopathic (majority of cases).
ii) Cases associated with systemic diseases (Hodgkin's
disease, HIV infection) and drug therapy (e.g. NSAIDs, rifampicin, interferon).
Nephrotic syndrome in MCD *in children* is characterized by *selective proteinuria* containing mainly albumin, and minimal amounts of high molecular weight proteins such as 2-macroglobulin. The basis for selective proteinuria appears to be as under:
i) Reduction of normal negative charge on GBM due to loss of heparan sulfate proteoglycan from the GBM.
ii) Change in the shape of epithelial cells producing foot process flattening due to reduction of sialoglycoprotein cell coat. *Adults* having MCD, however, have *non-selective proteinuria*, suggesting more extensive membrane permeability defect.

Clinical features. The classical presentation of MCD is of fully-developed nephrotic syndrome with massive and *highly selective proteinuria*, but hypertension is unusual. Most frequently, the patients are children under 16 years (peak incidence at 6-8 years of age).

HENOCH-SCHÖNLEIN PURPURA.

Henoch-Schönlein or anaphylactoid purpura is a self-limited type of hypersensitivity vasculitis occurring in children and young adults. Circulating immune complexes are deposited in the vessel wall consisting of IgA, C3 and fibrin, and in some cases, properdin suggesting activation of alternate complement pathway as the trigger event. The hypersensitivity vasculitis produces purpuric rash on the extensor surfaces of arms, legs and on the buttocks, as well as haematuria, colicky abdominal pain due to bleeding into the GIT, polyarthralgia and acute nephritis.

BENIGN NEPHROSCLEROSIS

Benign nephrosclerosis is the term used to describe the kidney of benign phase of hypertension. Mild benign nephrosclerosis is the most common form of renal disease in persons over 60 years of age but its severity increases in the presence of hypertension and diabetes mellitus. An important and early clinical marker for renal injury from hypertension and risk factor for cardiovascular disease is *macroalbuminuria* (i.e. Albuminuria > 150 mg/day or random urine albumin/creatinine ratio of >300 mg/gm creatinine), or *microalbuminuria* estimated by radioimmunoassay (i.e. microalbumin 30-300 smg/day or random urine microalbumin/creatinine ratio of 30-300 mg/gm creatinine). Currently, protein is measured in the urine with a 2-, 12-, or 24-hour urine collection, and spot checks of random urine samples using dipsticks that measure protein, creatinine, and protein ratios, or creatinine and albumin ratios. There are proteinuria tests that are promising, such as a cartridge-based automated analyzer.

Dipstick microalbumin:creatinine ratio testing has been suggested, but a review found that their use did not improve overall detection rates compared with automated or visual testing. Protein dipstick tests Point-of-care dipstick urinalysis for detection of proteinuria is the standard of care in most low-resource settings. However, urinary protein dipsticks have been shown to have low sensitivity and low specificity for urinary protein excretion over 24 hours.A prospective study found that the false-positive dipstick tests ranged from 7% at the 3+ level to 71% at the 1+ proteinuria level while false-negative rates were 7% for “nil” and 14% for “trace” proteinuria. Dipstick proteinuria was, however, significantly more likely to be correct (true positive/true negative when compared with 24-hour collection)

VISUAL INTERPRETATION OF URINARY DIPSTICK : Urinary dipsticks may have up to 10 chemical pads for measuring different substances in urine, including protein and albumin, although strips that restrict measurement to proteinuria or albuminuria are available. The advantage of a strip with multiple pads is that it can reveal associated urinary abnormalities that are causes of low-level proteinuria, such as haematuria or either asymptomatic bacteriuria or symptomatic urinary tract infection (both of which should be treated with antibiotics) by showing leukocytes and nitrites. The disadvantages include multiple results that may result in confusion and inappropriate further investigation; for example, leukocytes may be a completely normal finding in pregnancy given contamination of the urine by vaginal discharge. The urinary dipstick strip should be immersed completely in a well-mixed sample of urine for a short period of time, then extracted from the container and the excess urine removed by either supporting the edge of the strip over the mouth of the container, or drying the edges of the strip on absorbent paper . The strip is then left to stand for the time necessary for the reaction to occur (usually 60 seconds, as specified by the strip manufacturer). For visual analysis, the colour on the 'proteinuria' pad is compared with the chromatic scale specific to that strip and provided by the manufacturer. For automated analyses, the machine will read out the result. Results are reported as negative, trace, 1+, 2+, 3+, or 4+ based on the concentration of proteinuria detected. Although the concentration for a given '+' may vary from one manufacturer to another (particularly at the 4+ stage), 1+ proteinuria usually reflects 0.3 g/L of proteinuria.

HEAT COAGULATION TEST : The heat coagulation test may be used in under-resourced settings as an alternative to dipstick testing or other methods (discussed below) that are unavailable or too costly. A test tube is filled to two-thirds with urine. A few drops of dilute acetic acid are added to make the urine sample acidic. The upper part of the test tube containing urine is heated (but not boiled) over a burner. The presence of protein is signified by the turbidity of the urine when the tube is placed in front of a typed sheet of paper according to a pre-specified chart . The lower part of the tube of urine acts as a control as that urine should remain clear . The heat coagulation test may be less sensitive 1+≥than visually interpreted urinary dipsticks (at level) for detecting 0.3 g protein, however, it has reported specificity that is more tha n 90%.

LINE OF TREATMENT

Siddha treatment is not only for removal of disease, but for the prevention and improving the body condition. This is said as

1. Kaappu (Prevention)
2. Neekkam (Treatment)
3. Niraivu (Restoration)

Siddha system has unequivocally stated that even during the time of conception, some defects creep into the fertilized embryo. The defects form the basis for the manifestation of certain constitutional diseases later on during the existence of the individual. Like that diseases can be caused due to some external factors like, life style or even due to unknown causes; the disease which occurs due to unknown causes are designated as diseases of idiopathic origin or hereditary disorders. In Siddha system such diseases are described as Kanma noikal.

1.Kaappu (Prevention)

பிணையல் விதி

"தீண்மை ரஞ்ஞோசி க்க வக்காமற்
வெண்ணெய் மொன்றைப் புரக்காமல் - உண்ணுதல்
நீர்சுருக்கி மோர்பெருக்கி நெய்யுருக்கி யஞ்சுவர் தம்
பேரைக்கிற் போமே பிணி"

- பதார்த்த குணசிந்தாமணி

For prevention of disease we have to follow a better life style, in Siddha system of medicine to get rid of disease “Therar pini anukavithi” states about the things that we should follow for well being.

2. Neekkam (Treatment)

The Three Uyir Thathus which are responsible for organization, regularization and integration of the bodily structures and their physiological functions are always kept in a state of equilibrium by word, thought, deed and food of the individual. The general aetiological factors for constitutional discomfort is said to be incompatible diet, mental and physical activities. So it is essential to know the disease and the cause for the onset of the disease, before treating the

patient , also the nature of the patient, the severity of illness, the season and time of the occurrence of the diseases must be observed

Clinical Management for Disease Condition

- Normalization of altered uyirthathukal
- Internal medicines
- Asanas
- Diet

PREVENTION AND MANAGEMENT ASPECTS TO AVOID PROTIENURIA

Prevention: normoalbuminuric patients

The basis for the prevention of diabetic nephropathy is the treatment of its known risk factors: hypertension, hyperglycemia, smoking, and dyslipidemia. These are also risk factors for cardiovascular disease and should be vigorously treated. Intensive blood glucose control Clinical trials have consistently demonstrated that HbA1c levels <7% are associated with decreased risk for clinical and structural manifestations of diabetic nephropathy in type 1 and type 2 diabetic patients. Intensive treatment of diabetes reduces the incidence of microalbuminuria by 39% intensive glycemic control also reduces the rate of development of micro- and macroalbuminuria . Therefore, intensive treatment of glycemia aiming at HbA1c <7% should be pursued as early as possible to prevent the development of microalbuminuria.

Intensive blood pressure control

Treatment of hypertension dramatically reduces the risk of cardiovascular and microvascular events in patients with diabetes. Hypertension is common in diabetic patients, even when renal involvement is not present. About 40% of type 1 and 70% of type 2 diabetic patients with normoalbuminuria have blood pressure levels >140/90 mmHg even a reduction from 154 to 144 mmHg on systolic blood pressure reduces the risk for the development of microalbuminuria by 29% .

Blood pressure targets for patients with diabetes are lower (130/80 mmHg) than those for patients without diabetes . In a HOT (Hypertension Optimal Treatment) study, a reduction of diastolic blood pressure from 85 to 81 mmHg resulted in a 50% reduction in the risk of cardiovascular events in diabetic but not nondiabetic patients .

Renin-angiotensin system blockade

The role of ACE inhibitors in the prevention of diabetic nephropathy in patients with type 1 diabetes has not been defined. The use of perindopril during 3 years in normotensive normoalbuminuric type 1 diabetic patients delayed the increase in albuminuria . In patients with type 2 diabetes, ACE inhibitors and ARBs both diminish the risk for diabetic nephropathy and reduce the occurrence of cardiovascular events . In a MICRO-HOPE (Heart Outcomes Prevention Evaluation) study , ACE (10 mg/day) decreased the risk of overt nephropathy by 24% and the risk of cardiovascular death in patients with type 2 diabetes who were >55 years of age with one additional cardiovascular risk factor by 37. Therefore, ACE inhibitors have been shown to be beneficial for reno- and cardioprotection in patients with type 2 diabetes.

The goal of treatment is to prevent the progression from micro- to macroalbuminuria, the decline of renal function in patients with macroalbuminuria, and the occurrence of cardiovascular events. The treatment principles are the same as those adopted for the prevention of diabetic nephropathy, although in this case multiple and more intensive strategies must be used.

In siddha each herb, mineral or metal is said to be having six tastes, they are sweet,salt, sour, bitter, astringent, pungent.And all these tastes are formed by the combination of any two among panchabhoothas.

Table-8 –Taste and 5 element combination

Sweet	Earth+Water
Sour	Earth+Fire
Salt	Water+Fire
Bitter	Air+Space
Pungent	Air+Fire
Astringent	Earth+Air

In Athi nurai neer the prime cause for causing nurai was pitham, which is composed of thee bhootha.So it will be better to avoid or to take in minimal quantity the food stuffs having sour, salt, pungent taste; and better avoid sweet taste since almost all patients were diabetic.

Adverse effect of taking increased amount of sweet taste includes increased appetite, indigestion and it is stated that it can even lead to diseases such as Piramegam, Kandamaalai; so it would be better if this taste is taken in minimal amount.

Sour taste will aggeravate pitha (**வாற்றி லை விருத்தி யலைச் செய்யும்**). Since pitha is the main causative factor for mega noikal better to avoid this taste which is having the property of increasing pitha. Increased intake of sour food will cause tiredness, dizziness, dryness of tongue, increased heat, skin diseases etc all the features are present in diabetic cases so better avoid this taste.

Salt taste when taken in excess amount it will cause dryness of tongue, that is also a feature in diabetic cases, so it good to take this taste in minimal quantity. Bitter taste is having the property of normalizing pitham and kabam **பித்தம் கபம் ஆியலையின் விகற்பத்தை சாந்தி செய்யும்** so while taking the food stuffs with bitter taste it may normalize pitha and kaba.Astringent taste will cure pitha kaba diseases so this taste can be taken in adequate amount. Pungent taste is the comination of Air and Fire element so it is better to reduce the intake of pungent food. So it can be summarized that in diabetic cases or patient with mega noi Astringent, Bitter, Sweet taste should be taken in adequate quantity and to reduce intake of Pungent, Salt, Sour tastes.

Nutritional Intervention of Patient with Diabetic Nephropathy

Diabetic nephropathy occurs in 20% to 40% of patients with diabetes, and is a most likely causes of end-stage renal disease reported worldwide. Continuous microalbuminuria (30-299 mg/day) appears in the early stages of diabetic nephropathy with diabetes, and it is an indicator of nephropathy onset and the development of cardiovascular disease . A patient who had a progression from microalbuminuria to macroalbuminuria (≥300 mg/day) has a high risk of developing end-stage renal disease in few years . According to a recent study, the deterioration of renal function could be delayed by proactive management at the initial diagnosis point; therefore, it is important to implement a personalized diet customized to individuals as soon as possible. A healthy diet is very important for diabetic nephropathy patients. As eating properly can help slow down it’s progression, while eating improperly may worsen the condition.

Protein For people with diabetic kidney disease, it is of great importance to control the consumption of protein strictly, as a low-protein diet can help lighten the strain of kidneys effectively. In general, the daily protein intake can be 0.6-0.8g/kg. However, form the perspective of nutrition, they also need to eat some high-quality protein foods like lean meat, fish, chicken, milk, egg white and so on

DOS

- Fish, lean meat, milk, egg, high energy low proien
- potato, sweet potato, pumpkin,
- Low lipid consumption: olive oil,peanut oil(mono unsaturated fatty acid)
- Soy protien

- **DONTS**
- High potassium eg:pickle, chicken powder, processed cans
- High uric acid food : Animal giblets, sea foods, dried fish, beans.
- fresh fruits and vegetables,can be takenit provide vitamins and minerals.

3.NIRAIVU (RESTORATION)

Patients needs good discussion and motivation and persuation to accept the eventuality of Pitha disease and prepare for a lifestyle that provides optimization of metabolic status. In suitable effective medicinal preparations have to be administered in the beginning itself to neutralize and eliminate this disease.

Siddhars aimed at bringing the three doshas in equilibrium in the treatment of disease.. Siddhars prescribed a minimum dosage initially and then increased the dose gradually. There are thousand preparations for Pitha as well as Megha diseases and for its complications found in various Siddha text books Kudineer, Chooranams, Ilahams, Parpam and Chendooram.

Siddha system lays a great importance on the observation of rules regarding diet in everyday life because the Siddha system has rightly realized, that the basic factor of the body is food.

8.MATERIALS AND METHODS

8.1 Study type:

Observational study

8.2 Study plan
Activity / observation:

1. Informed written consent	:	On Day 0
2. Demographic Data	:	On Day 0
3. History taking	:	On Day 0
4. Physical examination	:	On Day 0
5. Laboratory investigations	:	On Day 0
6. Inclusion / Exclusion criteria	:	On Day 0
7. Performing Neikkuri	:	On Day 1, 2, 3
8. Documentation	:	On Day 1, 2, 3

8.3 Study place:

- OPD & IPD,
  Ayothidoss Pandithar hospital,
  National Institute of Siddha,
  Chennai- 47.

8.4. Population and sample:

- 18-70 age groups fulfilling all the inclusion criteria and passing the exclusion criteria mentioned below.
- The sample consists of patients attending the OPD & IPD of Ayothidoss Pandithar Hospital, National Institute of Siddha.

8.5. Sample size:

- Total : 60

  Athinurai neer (Albuminuria) patients : 30
  Healthy Volunteers : 30

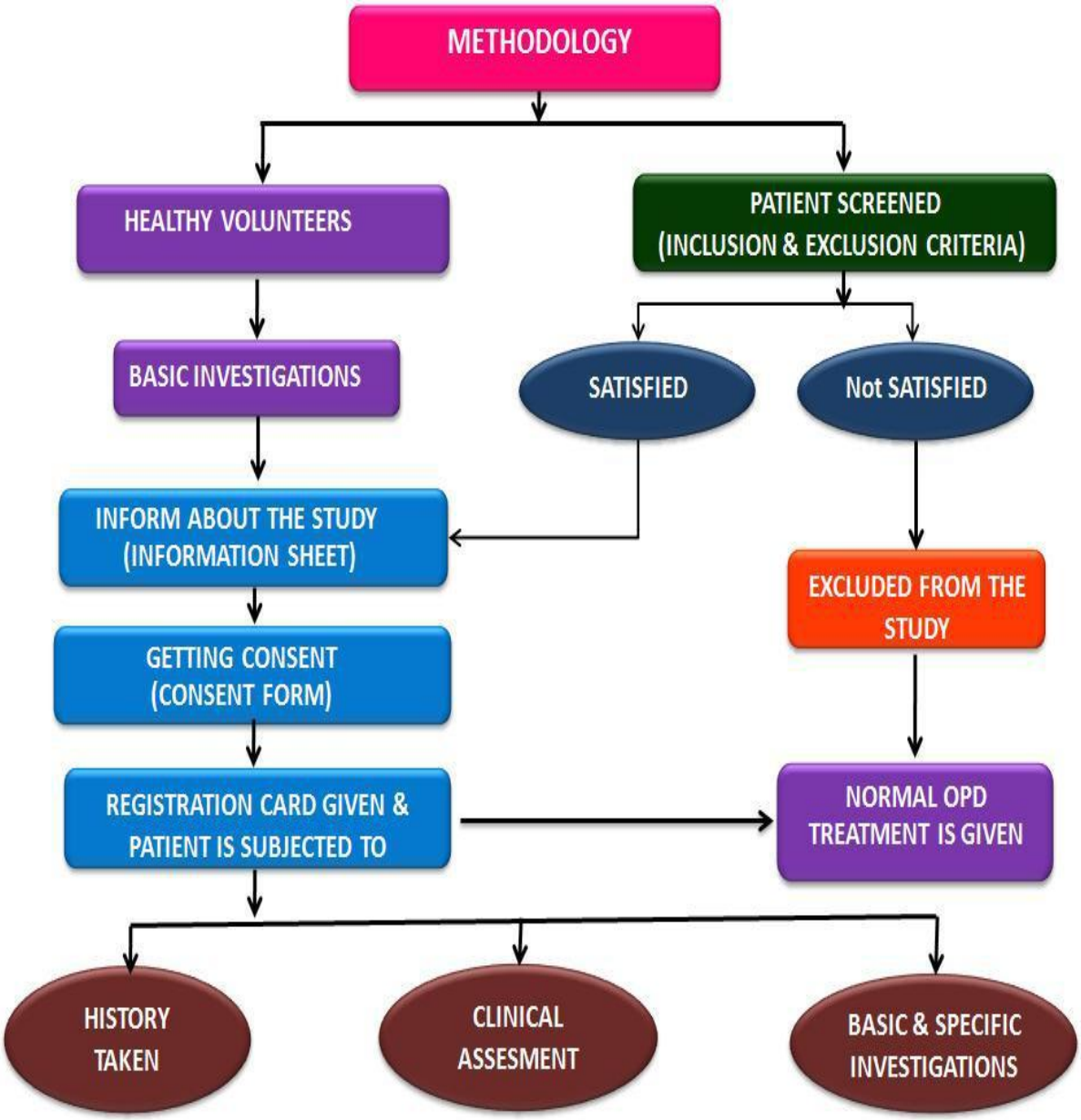
8.6. Selection criteria

8.7 Inclusion criteria:

- Age:18-70 Years
- Patients who had urine albumin positive with increased froth in different examinations .

8.8. Exclusion criteria:

- Patients who were undergoing dialysis.
- Patients with high fever
- Patients with serious systemic illness



8.9 Investigations Blood

- TC
- DC
- ESR
- Hb
- FBS
- PPBS
- S. Cholesterol

Urine

- Albumin
- Sugar
- Deposits

Motion

- Ova
- Cyst
- Occult blood

Establishing the diagnostic characteristics of Athi Nurai Neer/Frothy urine (Albuminuria condition) throughEight fold examination (8+2)

Naadi

- Naadi nithanam
- Naadi nadai

Meikuri (Physical Signs)

- Veppam
- Viyarvai
- Thodu vali

Naa (Tongue)

- Maa padithal
- Niram
- suvai
- Vaineer ooral
- Vedippu

Niram (Complexion)

- Karuppu
- Manjal
- Veluppu

Mozhi (Voice)

- Sama oli,
- Urattha oli,
- Thazhntha oli

Vizhi (Eyes)

- Niram
- Kanneer vadithal
- Erichal
- Peelai seruthal

Malam(Stools)

- Niram
- Sikkal
- Kalichal
- Sirutthal
- Seetham

Moothiram (Urine)

Neerkuri

- Niram
- Manam
- Edai
- Nurai
- Enjal

Neikuri
Manikkadainool

8.10 Neikkuri procedure:

Source of oil: Oil was procured in a mill from freshly ground gingely seeds in stone grinder (chekku)without any additives being added to avoid variations in the reactions**.** Because the presently marketed Gingely oils are treated with additives for which reason I chose the above method of additive free preparation**.**

Bowl –Porcelain bowl

Structure of the bowl:

Base- flat

Mouth –wide

Neck –Diverged

Method of oil instilling:

- Distance between the bowl & the oil stick was 3-4 cm.
- Below 3cm, the stick may inadvertently touch the bowl, above 4cm, the oil may be dispersed due to air or it may cause ripples over the surface of the urine sample interfering with the results of the examination.

Diet pattern:

- Quality- balanced food with appropriate proportion of all six tastes
- Quantity- upto the level of his appetite

Sleep pattern

- Sound sleep

Collection of urine

- Time period –early morning (4am-7am) for IP & OP patients
- After the collection of urine sample, the neikkuri was performed within one and half hour.

Neikkuri picture:

(Photo documentation with standard Digital imaging) fourslides of picture will be take

1. At the moment after dropping of oil.
2. At 1 minute .
3. At 3 minute.
4. At 10 minute.

Procedure

Collected urine sample for Neikkuri in a sterile porcelain bowl. Then instilled a drop of gingely oil using a stick and observe the nature of spreading of oil in urine for 10 minutes.

- Photo documentation with standard digital imaging.
- Complete urine analysis.
- The above Neikkuri procedure was repeated (except urine analysis) for next two consecutive days.

8.11. Data collection

Case Record Form
Annexure I : Screening and selection proforma
Annexure IA : History proforma
Annexure II : Clinical Assessment Form
Annexure III : Laboratory Investigations
Aannexure IV : Informed Written Consent Form
Annexure IVA: Patient Information Sheet

8.12.Data management

After enrolling the patient in the study, a separate file for each patient was opened and all forms were filled in the file. Study no, and Patient no was entered on the top of the file for easy identification and arranged in separate rack at the concerned OPD unit. Whenever study patient visits OPD during the study period, the respective patient file was taken and necessary recordings were made at the case recording form or other suitable form.

The data recordings were monitored for complication and compliance of patient by HOD and Sr. Research Officer (Statistics). All forms were further scrutinized in presence of investigators by Sr. Research Officer (Statistics) for logical errors and incompleteness of the data before entering on the computer to avoid any bios. No modification in the results was permitted for unbiased report.

Any missed data found in during the study, it was collected from the patient, but the time related data was not record retrospectively. All collected data were entered using MS access software onto computer. Investigators were trained into enter the patient data and cross checked by SRO]

8.13.Statistical analysis

All collected data were entered into computer and the neikuri shape was recorded as per literature. The shape association with Normal healthy individuals / in patients with Athi Nurai Neer were descriptively analyzed and presented.

8.14.Ethics Issues addressed

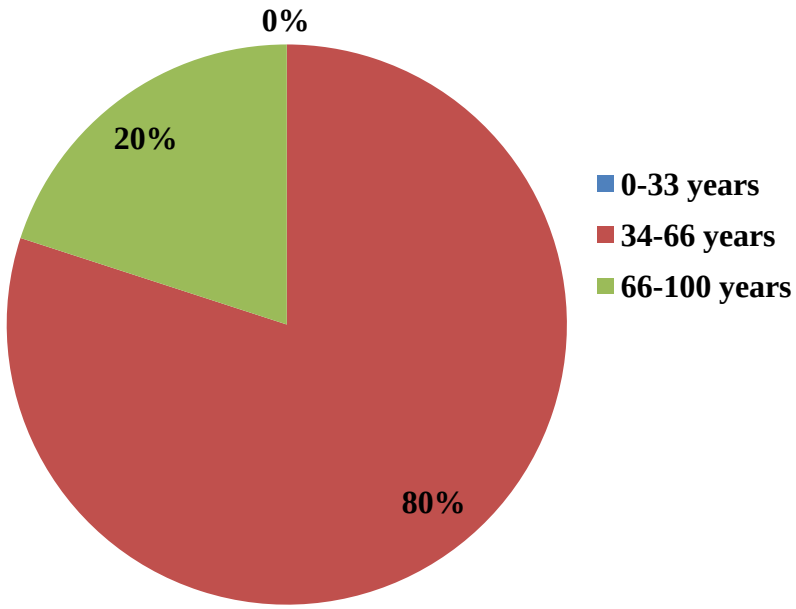
- Patients were examined and screened in an unbiased manner and were subjected to the criteria.
- Informed consent were obtained from the patient in writing, explaining in the understandable language to the patient.
- The data collected from the patient were kept confidential. The study patient were explained about the diagnosis.
- To prevent any infection, while collecting blood sample from the patient, only disposable syringes, disposable gloves, with proper sterilization of lab equipments were used.
- This study involved only the necessary investigations (mentioned in the protocol) and No other investigation were done.
- Normal treatment procedure followed in NIS was prescribed to the study patients and the treatment was provided free of cost.
- There was no infringement on the rights of patient

9.1 AGE DISTRIBUTION

Table-9 Age distribution

Sl No	Age Distribution	No of cases	Percentage
1	0-33 years	0	0
2	34-66 years	24	80
3	66-100 years	6	20
4	Total	30	100

Fig 1 Age distribution



Observation

Among 30 cases 24 cases that is 80% belonged to the category of age group 34-66 years, 0nly 20% belonged to 66-100 years category. None belonged to 0-33 category.

Inference

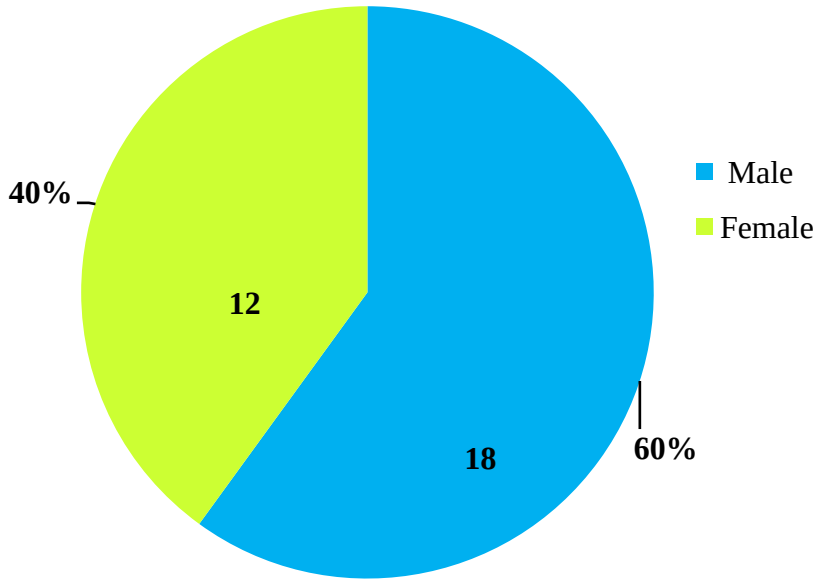
80 % of the cases belonged to the age category 34-66 years.

9.2 SEX DETERMINATION

Table-10 Sex determination

Sl No	Sex determination	No.of cases	Percentage
1	Male	18	60
2	Female	12	40
3	Total	30	100

Figure 2 – Sex deermination



Observation

Among thirty cases 18 cases that is 40% of cases were females and 12 cases that is 60% of cases were male.

Inference

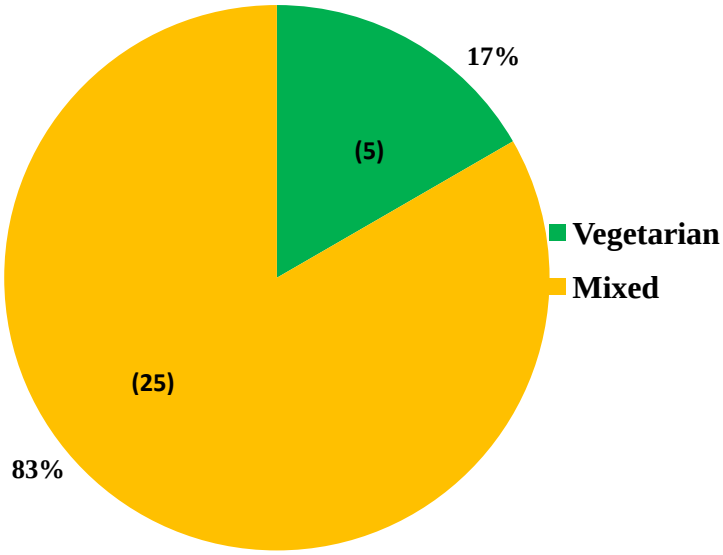
60 % of selected cases were males, as per studies prevalence of diabetes is equal in men as well as women, but in this hospital based study since the sample size was limited it showed more incidence in males when compared to female.

9.3 FOOD HABITS

Table-11 Food habits

Sl No	Food habits	No of cases	Percentage
1	Vegetarian	5	16.66
2	Mixed	25	83.33
3	Total	30	100

Figure 3- Food habits



Observation

Among thirty cases 25 cases were having mixed diet, that is (83%) and 5 cases were vegetarians that is (17%).

Inference

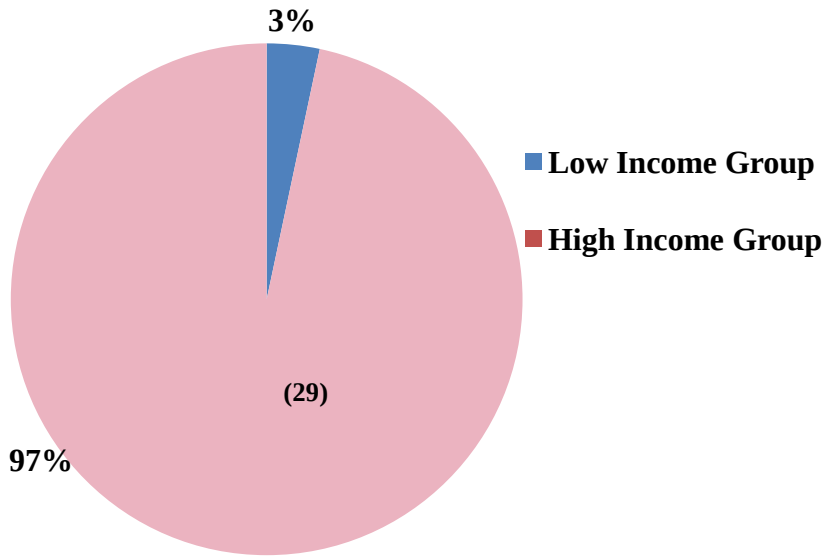
Most of the cases were being non-vegetarian. Non-vegetarian diet which is considered as thamo gunam food seem to alter the normal constitution of a person and cause disease.

9.4 SOCIO- ECONOMIC STATUS

Table-12 – Socio-economic status

Sl No	Economic status	No.of cases	Percentage
1	Low Income Group	1	3.33
2	High Income Group	0	0
3	Middle Income Group	29	96.66
4	Total	30	100

Figure 4- Socio- Economic status



Observation

Among thirty cases 29 cases (96.66 %) were under middle income group and only one case (3.33%) was low income.

Inference

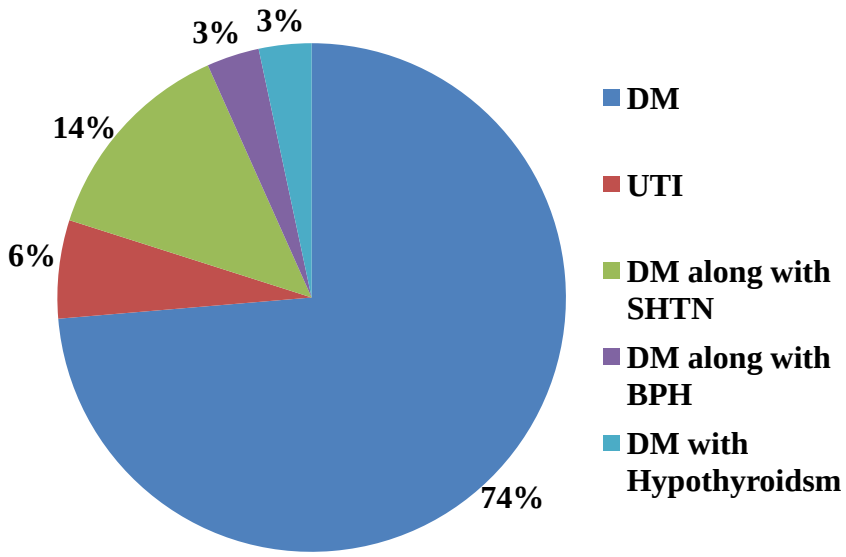
Majority of cases belong to middle income group. As per WHO report of 2016 , an estimated 347 million people in the world had diabetes and the prevalence is growing particularly in low- and middle income counties; as per this study all the cases came under socio economic status of either low income or middle income group.

9.5 ETIOLOGY OF NURAINEER

Table-13 Etiology of nurainеer

Sl No	Etiology	No of cases	Percentage
1	DM	22	73.33
2	UTI	2	6.22
3	DM along with SHTN	4	13.33
4	DM along with BPH	1	3.33
5	DM with Hypothyroidsm	1	3.33

Fig.5 Etiology



Observation

Among thirty cases 28 cases (78%) were Diabetic, 2 cases were having urinary tract infection, 1 patient had diabetic along with BPH, and one another had diabetic along with hypothyroidism, 4 cases had diabetic along with hypertension.

Inference

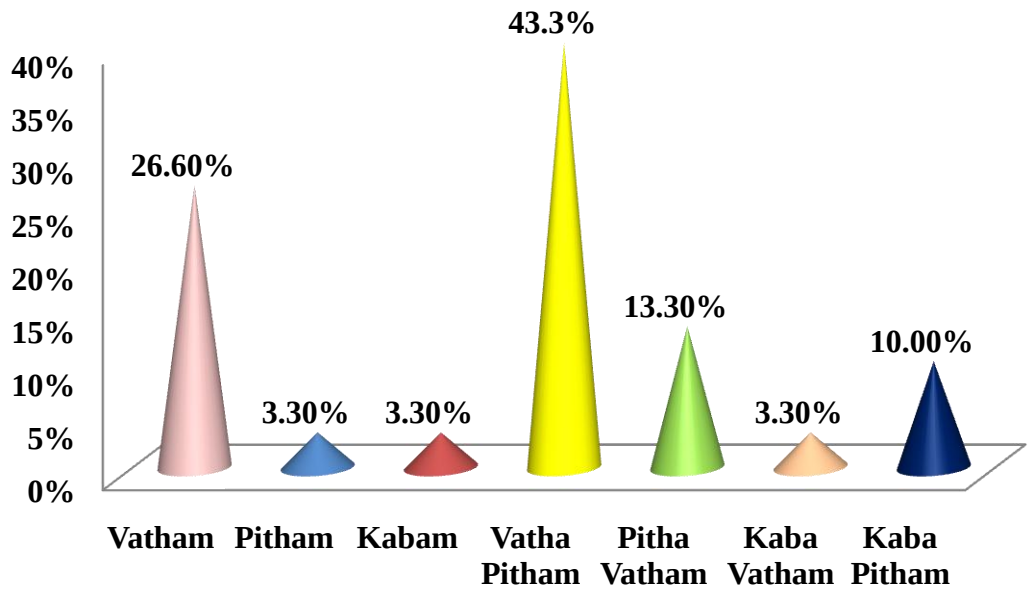
Majority of cases were with Diabetic etiology, so the most common cause for Nurai neer (Albuminuria) was Diabetes Mellitus in the selected subjects who came to NIS.

9.6 YAKKAI ELAKKANAM

Table-14 Yaakkai ilakkanam

Sl No	Yakkai	No of cases	Percentage
1	Vatham	8	26.6
2	Pitham	1	3.3
3	Kabam	1	3.3
4	Vatha Pitham	13	43.3
5	Pitha Vatham	4	13.3
6	Kaba Vatham	1	3.3
7	Kaba Pitham	2	6.6

Figure 6 – Yaakai Ilakkanam



Observation

Among thirty cases 13 patients (43.3%) were having Vatha-pitha temperament, 8 (26.6%) had Vatha temperament, 4(13.3%) had Pitha vatha temperament 3 (10%) had Kaba pitha temperament. Only single cases had Pitha, Kaba and Kaba Vatha temperament.

Inference

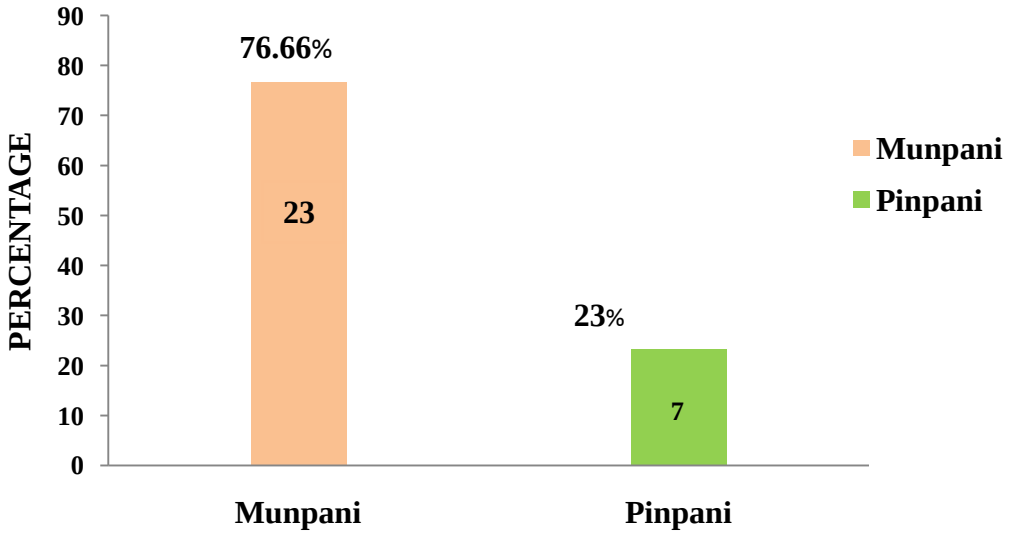
Majority of cases were having Vatha Pitha udalinan(temperament). Patients with Vatha pitha temperament subjectsnwould generally be lean in nature, darker complexion, majority of cases did not have these features since the sample size was limited it was not able to substantiate the literature.

9.7 NOI UTRA KAALAM

Table-15 Noi utra kaalam

Sl No	Noi utra kaalam	No of cases	Percentage
1	Munpani	23	76.66
2	Pinpani	7	23.33

Figure 7 – Noi utra kaalam



Observation

Out of 30 cases 23 cases (76.66%) were had affected in Munpani kaalam and 7 cases (23.3%) cases had affected in Pin pani kaalam.

Inference

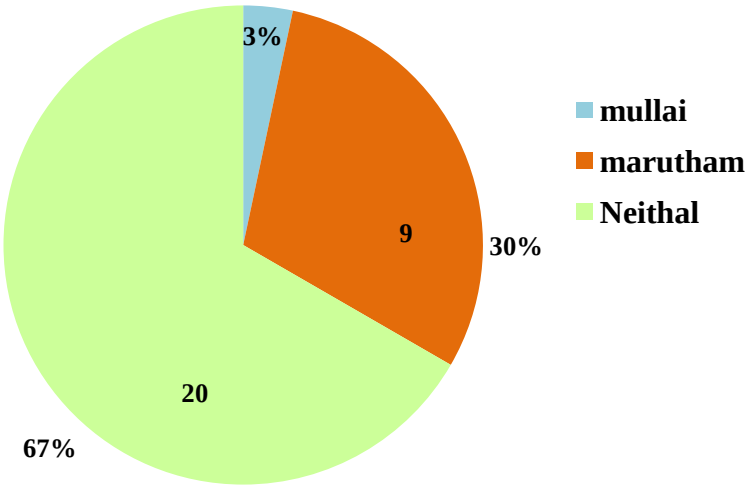
Majority of cases in the study got affected with the disease during Munpani kaalam(Margazhi, Thai)

9.8 NOI UTRA NILAM

Table-16- Noi utra nilam

Sl No	Noi utra nilam	No of cases	Percentage
1	Mullai	1	3.33%
2	Maruthvam	9	30%
3	Neithal	20	66.66

Figure 8 – Noi utra kaalam



Observation

Out of 30 cases 9 (30%) patients were from Marutham nilam, and 1(3%) were from Mullai nilam.20 cases (67%) were from Neithal nilam

Inference

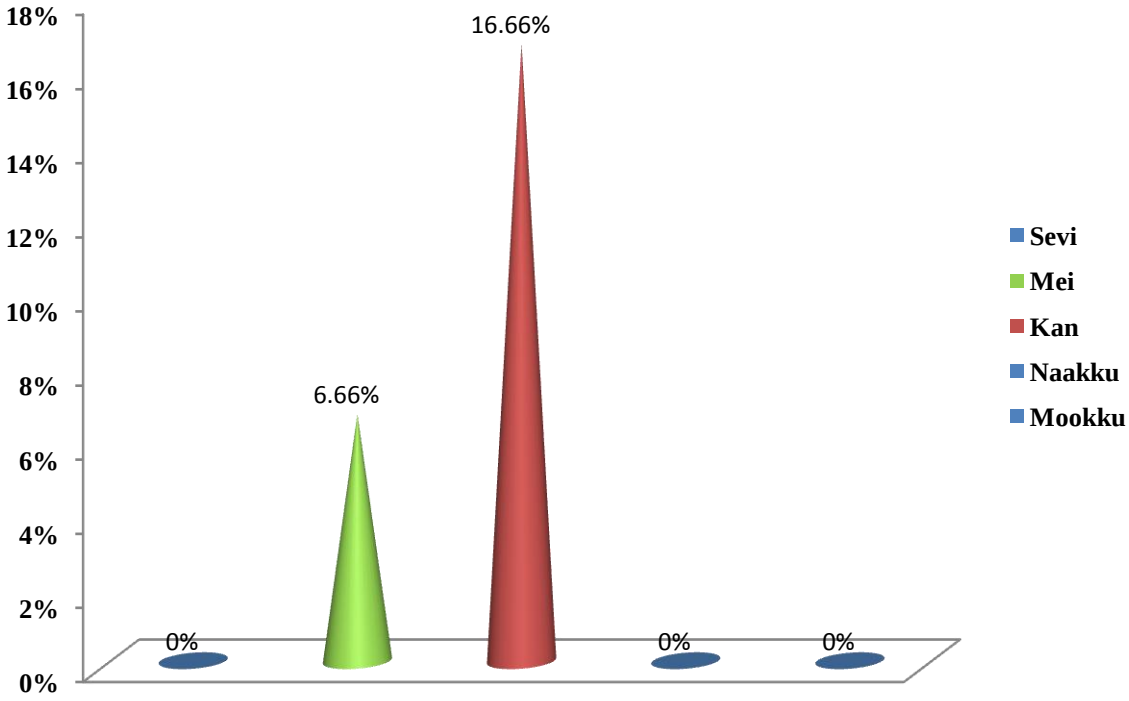
Majority of cases hailed from Neithal Nilam.

9.9 GNANENTHIRIYANGAL

Table-17 Gnanenthiriyangal

Gnanenthiriyangal			
Sl No	**Gnanenthiriyangal**	**No of cases**	**Percentage**
1	Sevi	0	0
2	Mei	2	6.66
3	Kan	5	16.66
4	Naakku	0	0
5	Mookku	0	0

Figure 9 –Gnanenthiriyangal



Observation

Out of 30 cases in 2 (6.66%) patients , Mei was affected and in 5 (16.66%) cases kan was affected.

Inference

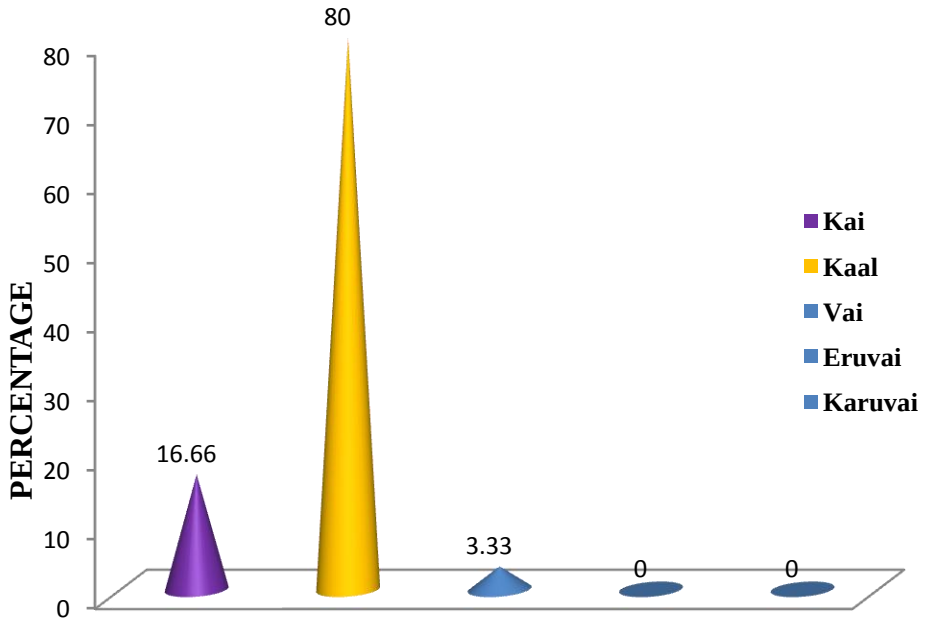
Only Mei and Kan were affected among Gnanenthiriyas in the patients with complaints of athi nurai neer.Out of 30 cases 23 subjects were having normal Gnanenthiriyangal. So as per this study gnanenthiriyam did not show much significance.

9.10 KANMENTHIRIYANGAL

Table-18 Kanmenthiriyangal

Kanmenthiriyangal			
Sl No	**Kanmenthiriyangal**	**No of cases**	**Percentage**
1	Kai	5	16.66
2	Kaal	24	80
3	Vai	1	3.33
4	Eruvai	0	0
5	Karuvai	0	0

Figure 10 - Kanmenthiriyangal



Observation

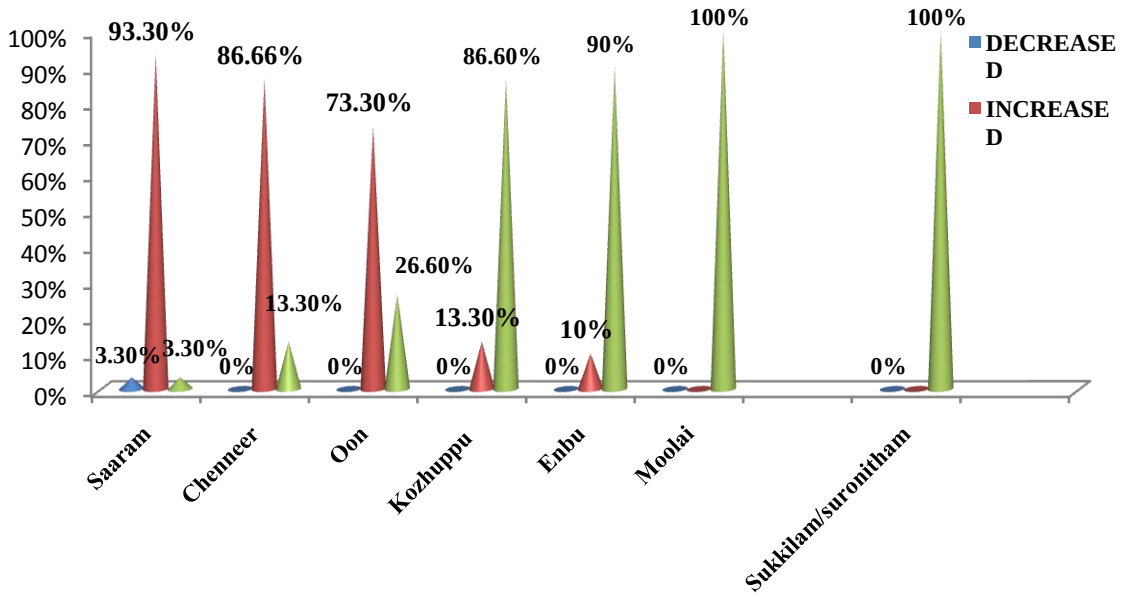
Out of 30 cases in 24 (80%) patients kaal was affected , kai was affected and in 5 (16.66%) cases , vaai was affected in 1 (3.33%)case.

Inference

Kai, kaal and vaai were affected in Athinurai neer cases. Kaal was affected in majority (80%) , it may be due to degenerative changes or diabetic neuropathy because majority of patients were diabetic and belonged to middle age group or old age group.

9.11 UDAL THAATHUKKAL

UDAL THAATHUKKAL									
Sl No	**Udal thathu**	**Increased**		**Decreased**		**Normal**		**Total**	
1	Saaram	1	3.33	28	93.33	1	3.33	30	100
2	Chenneer	0	0	26	86.66	4	13.33	30	100
3	Oon	0	0	22	73.33	8	26.66	30	100
4	Kozhuppu	0	0	4	13.33	26	86.66	30	100
5	Enbu	0	0	3	10	27	90	30	100
6	Moolai	0	0	0	0	30	100	30	100
7	Sukkilam/suronitham	0	0	0	0	30	100	30	100



Observation

Out of 30 cases in 1 case (3.33%) saaram was increased, in 28 cases (93.33%) saaram was decreased. Chenneer was decreased in 26 cases (86.66), 4 cases (13.33%) were normal. Oon was decreased in 22 cases (73.33%) and normal in 8 cases (26.66%). Kozhuppu was decreased in 4 cases (13.33%), normal in 26 cases (86.66%).Enbu was decreased in 3 cases (10%)and normal in 27 cases (90%).Moolai, Sukkilam and Suronitham was affected in none of the cases.

Inference

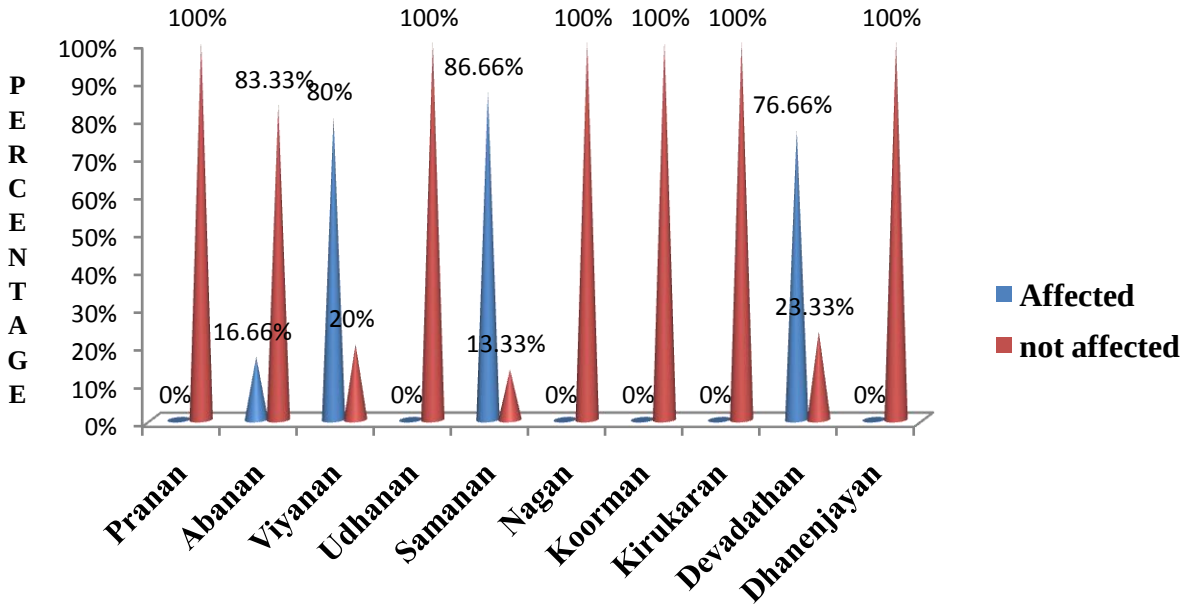
Saaram , chenneer, oon, kozhuppu was decreased in majority of cases, among udal kattukkaal saaram was increased in only one case. Enbu , Moolai, Sukkilam and Suronitham was not affected in any of the cases. Majority of cases subjected to study were diabetic, as per siddha literatures udal thathukkal will get affected one by one in Madhumegham ; here saaram , chenneer, oon, kozhuppu were affected in majority of cases justifying the literature.

9.12 UYIR THATHUKKAL – VALI

Ta ble-20 Uyir Thathukkal-Vali

	Vali					
Sl no		**Affected**	**Percentage**	**Not affected**	**Percentage**	**Total**
1	Pranan	0	0	30	100	30
2	Abanan	5	16.66	25	83.33	30
3	Viyanan	24	80	6	20	30
4	Udhanan	0	0	30	100	30
5	Samanan	26	86.66	4	13.33	30
6	Nagan	0	0	30	100	30
7	Koorman	0	0	30	100	30
8	Kirukaran	0	0	30	100	30
9	Devadathan	23	76.66	7	23.33	30
10	Dhanenjayan	0	0	30	100	30

Figure 11 Uyir Thathukkal - Vali



Observation

Out of 30 cases Pranan was not affected in any of the cases, Abanan was affected in 5 (16.66%) cases, Viyanan was affected in 24 (80%) cases, Udhanan, Naagan, Koorman, Kirukaran, Dhanenjayan was affected in none of the cases, Samanan was affected in 26(86.66%) cases, Devadathan was affected in 23 (76.66%) cases.

Inference

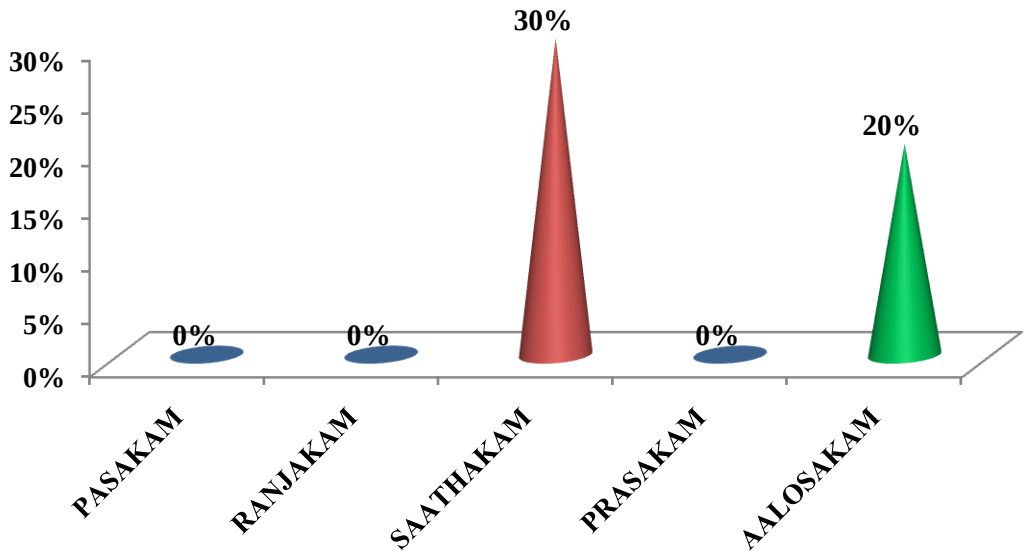
Viyanan, Devadathan and Samanan were affected in majority of cases, Abanan was affected in five and other vaayus was not affected in any of the cases. Fatigue is common in diabetes which is a feature of devaduthan, hence it shows significance in the study.

9.13 **UYIR THATHUKKAL- AZHAL**

Table 21– Uyir Thathukkal- Azhal

Pitham			
Sl no	**Azhal**	**No of cases**	**Percentage**
1	Pasakam	0	0
2	Ranjakam	0	0
3	Saathakam	9	30
4	Prasakam	0	0
5	Aalosakam	6	20

Figure 13– Uyir thathukkal Azhal



Observation

Out of 30 cases Saathagam was affected in 9 (30%) cases, Aalosakam was affected in 6 (20%) cases.Pasakam, Ranjakam, Prasakam was not affected in none of the cases.

Inference

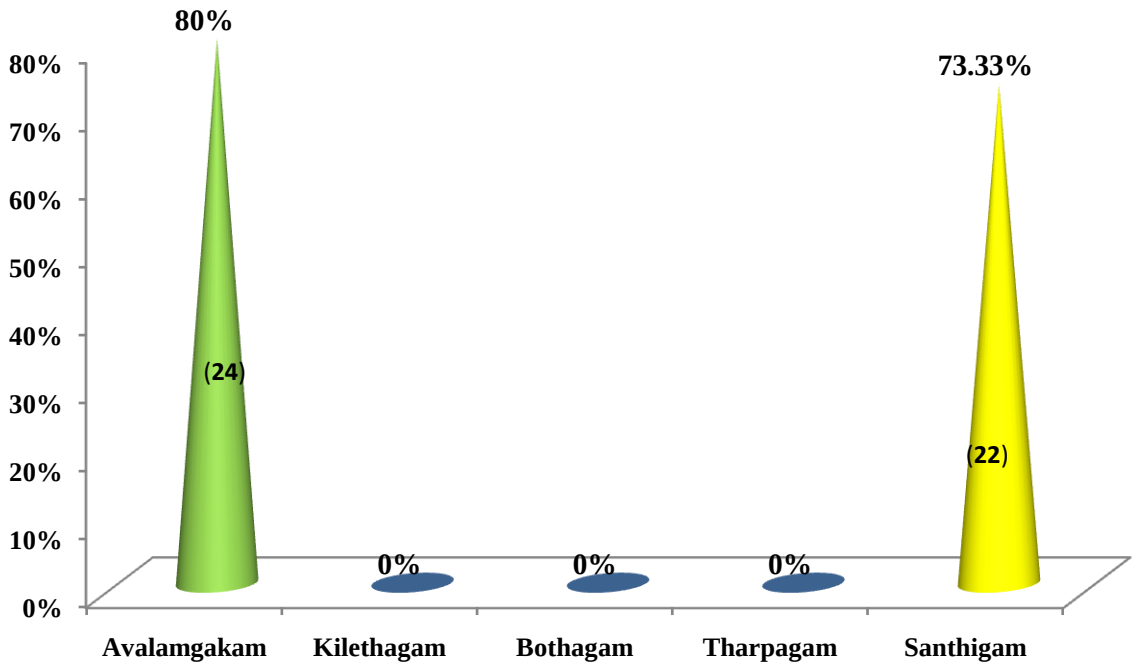
Aalosakam, Saathagam were the affected components of Pitham, any diseased one will be having difficulty in doing desired activities, which is a feature of saathagam.

9.14 **UYIR THATHUKKAL- IYYAM**

Table 22– Uyir Thathukkal- Iyyam

IYYAM			
SL NO	**Iyyam**	**No of cases**	**Percentage**
1	Avalamgakam	24	80
2	Kilethagam	0	0
3	Bothagam	0	0
4	Tharpagam	0	0
5	Santhigam	22	73.33

Figure 14– Uyir thathukkal Iyyam



Observation

Out of 30 cases Avalambakam was affected in 24 cases (80%), santhigam was affected in 22(73.33%). None among the components other than avalambakam and santhigam were affected in any of the cases.

Inference

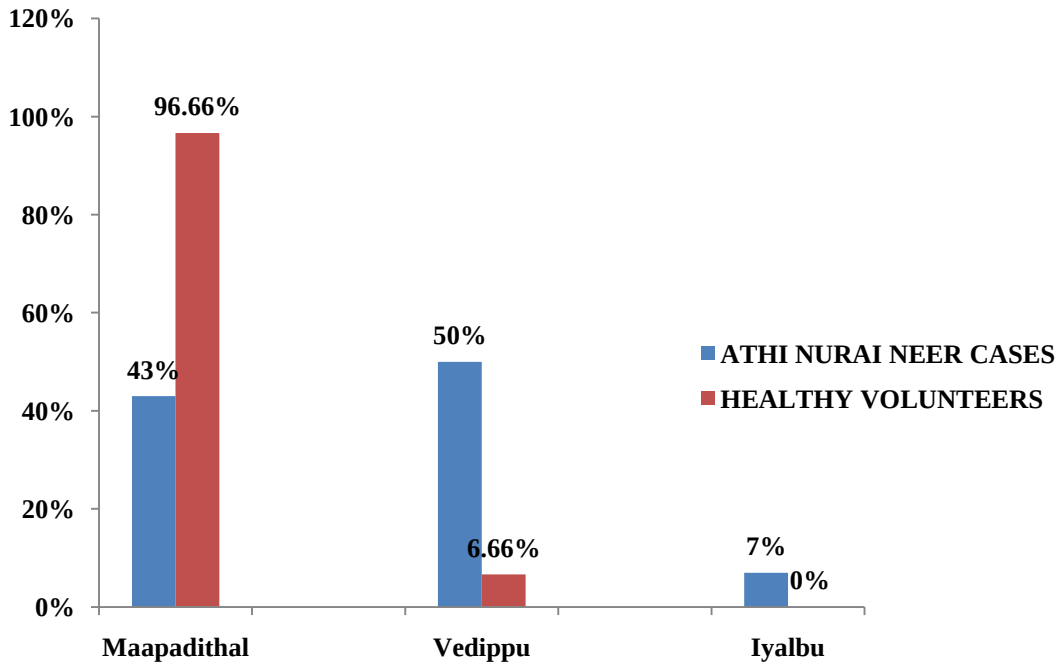
Avalambakam and santhigam were the only affected components of kabam among the selected subjects with complaints of Athi Nurai Neer who came to NIS.

9.15 ENVAGAI THERVUGAL

Table -23 Envagai thervugal

Naa		Athi nurai neer		No.of. Healthy Volunteers	Percentage
		No.of cases	Percentage		
Thanmai	Maapadithal	13	43	29	96.66
	Vedippu	15	50	2	6.66
	Iyalbu	2	7	0	0
	Total	**30**	**100**	**30**	**100**
Niram	Karuppu	2	6.66	6	20
	Manjal	24	80	19	57.57
	Veluppu	4	13.33	5	16.66
	Total	**30**	**100**	**30**	**100**
Suvai	Kaippu	1	3.33	0	0
	Pulippu	1	3.33	1	3.33
	Inippu	2	6.66	8	26.66
	Normal	26	**86**	**21**	**70**
	Total	30	100	30	100
Vaineerooral	Increased	1	3.33	1	3.33
	Iyalbu	29	96.66	29	96.66
	Total	**30**	**100**	**30**	**100**

Figure 15- Naa thanmai



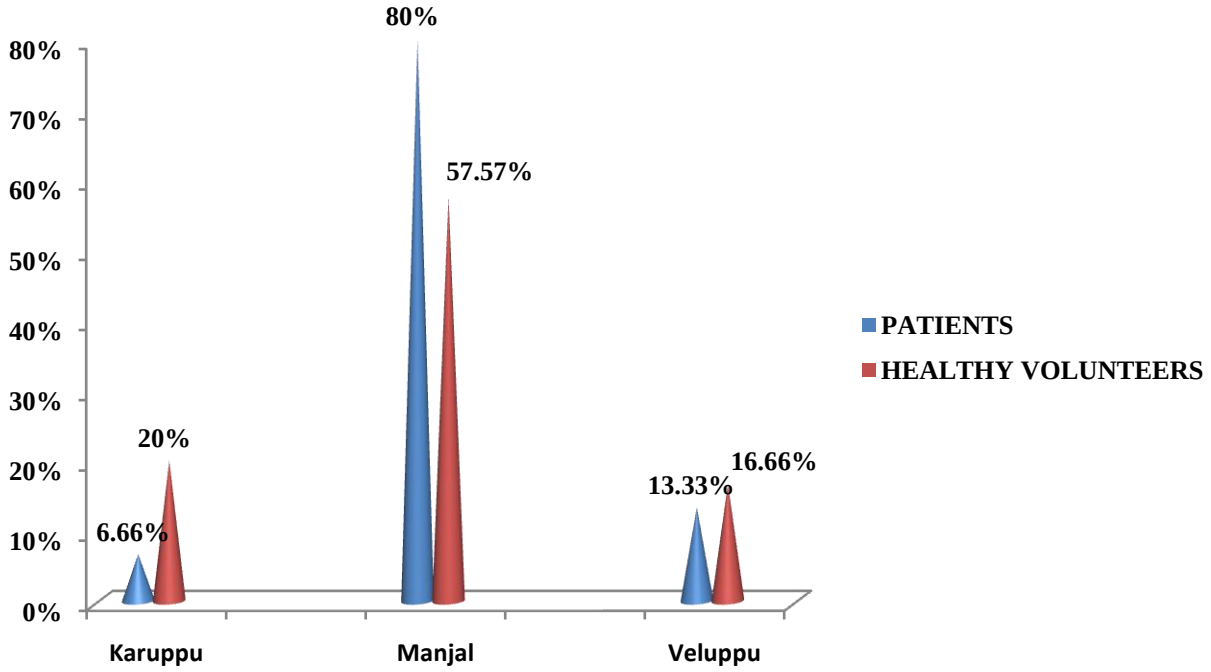
Observation

Out of 30 patients when tongue was observed, Maa padithal was seen in 13 (43%) , in case of healthy volunteers 29 (96.66%) had maa padithal over tongue. Vedippu was present in 15(50%) patients, 2 (6.66%) healthy volunteers.2(7%) among 30 patients were normal and none of the healthy volunteers had normal tongue.

Inference

As per this study none of the healthy volunteers had a normal tongue. All healthy volunteers had either maa padithal or vedippu.

Figure 16- Colour of tongue



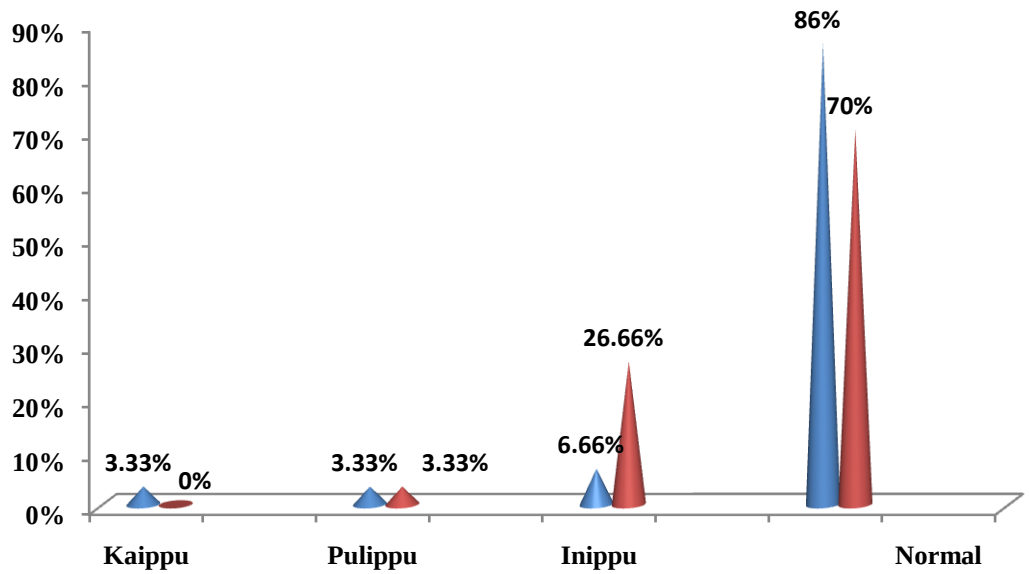
Observation

Out of 30 cases when colour of tongue was observed, Karuppu (black) colour was seen in 2 (6.66%) patients, and 6 (20%) healthy volunteers; manjal(yellow) colour was seen in 24 (80%) patients; veluppu (white) colour was observed in 4 (13.33%) patients,5(16,66%) healthy volunteers.

Inference

There was no significance in colour observation of tongue because blackish discolouration and whitish discolouration was shown more by healthy volunteers when compared to patient. Yellowish discolouration of tongue was shown in a higher percentage 80% in patients and 57.57% in healthy volunteers, this may be an indication of deranged pitham; but this doesn't seems to be much significant according to this study.

Figure 17 – Taste in tongue



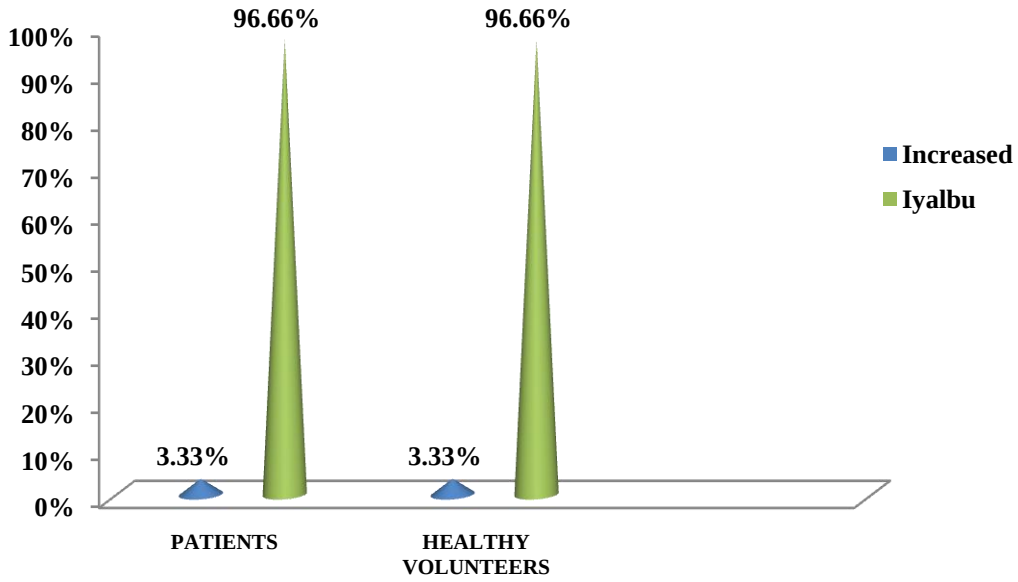
Observation

Out of thirty patients 1(3.33%) had Kaippu (Bitter taste) in tongue, none of the healthy volunteers had bitter taste in tongue; pulippu(sour) 1(3.33%) on ; each both patients and healthy volunteers; inippu(sweet) in 2 (6.665) patients and 8(26.66%) healthy volunteers; 26(86%) out of 30 patients had normal taste sensation, 21 (70%) among healthy volunteers had normal taste sensation in tongue.

Inference

Majority of normal subjects as well as patients had normal taste sensation .Even though sweet taste sensation of tongue is present in diabetic patients this was not significantly observed in patients subjected to study.

Figure 18 - Salivation



Observation

Vai neer ooral (salivation) was normal in 29 out of 30 cases both in patients and healthy volunteers.One on each had hypersalivation.

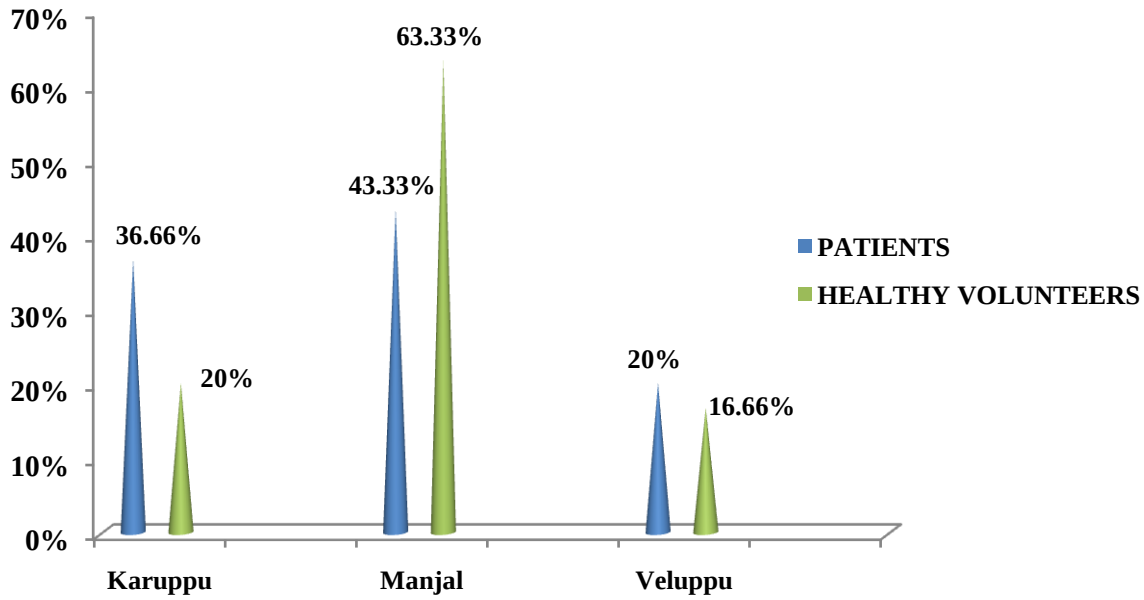
Inference

Salivation was not a significant observation because one on each that is patients and healthy volunteers had hypersalivation.

Table 24- Niram, Mozhi and Vizhi

Niram, Mozhi and Vizhi		Athi nurai neer		No.of. Healthy volunteers	Percent Age
		No.of. cases	Percent Age		
Niram	Karuppu	11	36.66	6	20
	Manjal	13	43.33	19	63.33
	Veluppu	6	20	5	16.66
	Total	**30**	**100**	**30**	**100**
Mozhi	Sama oli	29	96.66	25	83.33
	Urattha oli	0	0	2	6.66
	Thazhntha oli	1	3.33	3	10
	Total	**30**	100	60	**100**
Vizhiyin Niram	Karuppu	0	0	0	0
	Manjal	14	**46.66**	**0**	0
	Sivappu	0	0	0	0
	Veluppu	15	50	1	3.33
	Iyalbu	1	3.33	29	96.66
	Total	**30**	**100**	**30**	**100**
Vizhiyin Thanmai	Kanneer	0	0	0	0
	Kan Erichchal	0	0	0	0
	Peelai seruthal	0	0	0	0
	Iyalbu	30	100	30	100
	Total	**30**	**30**	**30**	**100**

Figure 19 - Complexion



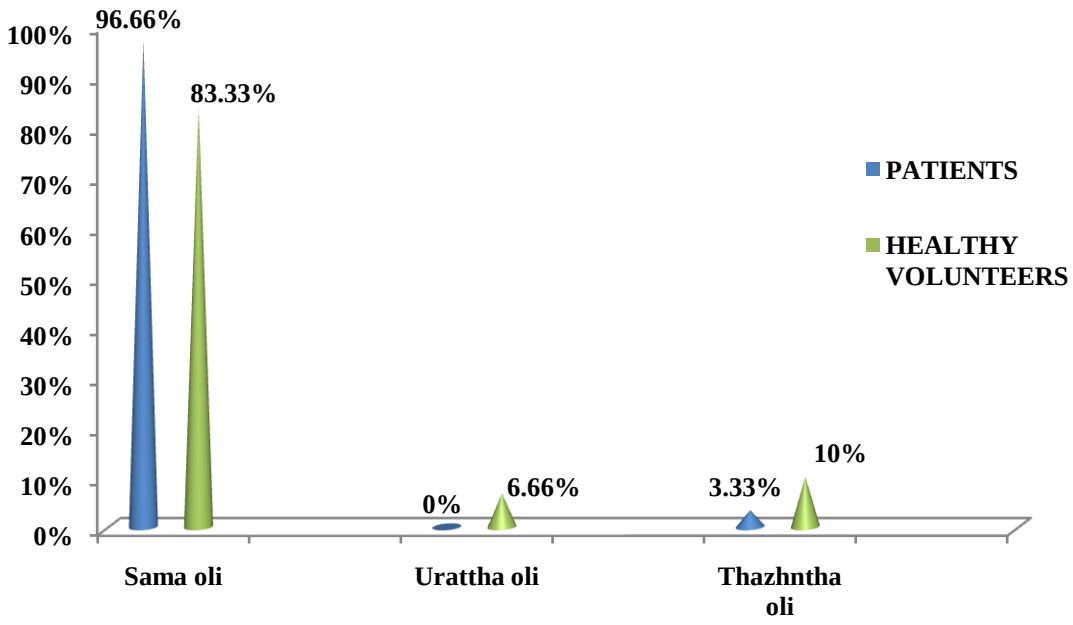
Observation

Out of 30 cases when complexion was observed dark complexion was seen in 11 (36.66%) patients and 6(20%) healthy volunteers; yellowish in 13(43.33%)patients, and 19 (63.33%) healthy volunteers; whitish in 6(20%) patients and 5 (16.66%) healthy volunteers.

Inference

Majority of patients as well as healthy volunteers had yellowish complexion, colour does not show much significance in this study.

Figure 20 - Mozhi



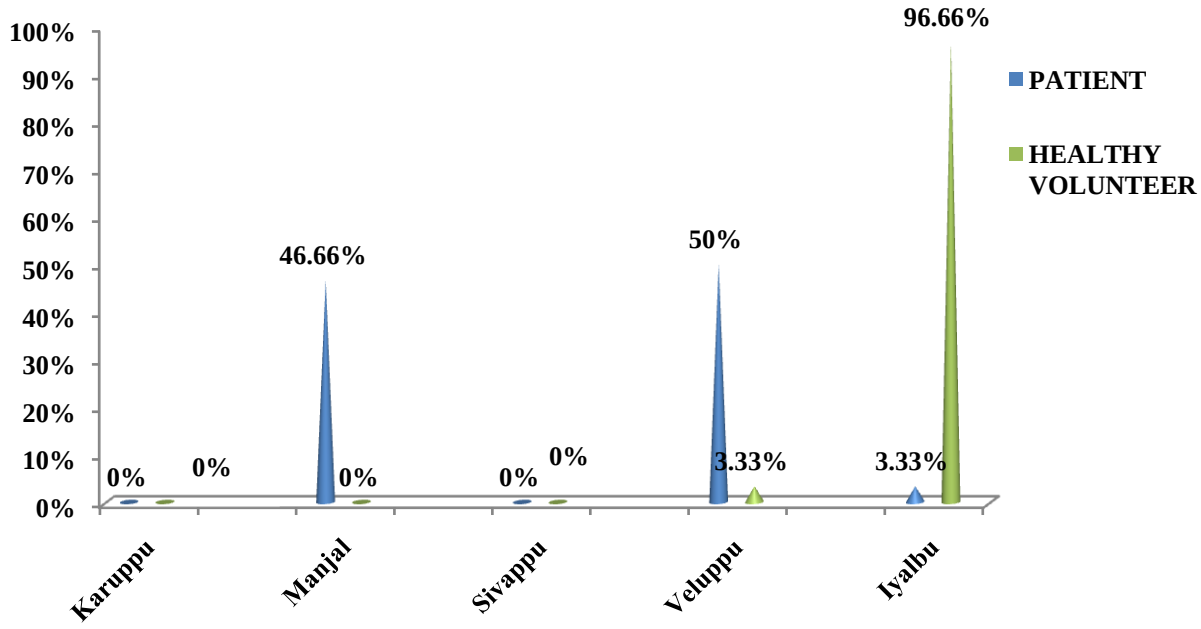
Observation

Out of 30 patients 29 (96.66%) had sama oli, 25 (83.33%) among healthy volunteers; none among the patients , 2 (6.66%) had uratha oli among healthy volunteers; one (3.33%)among patient and 3(10%) among healthy volunteers had thazhntha oli.

Inference

Majority of patients as well as healthy volunteers had sama oli.

Figure 21 – Vizhiyin niram



Observation

Out of 30 cases none had blackish discolouration of eyes, yellowish discolouration was seen in14 (46.66%) patients and none among healthy volunteers. Reddish discolouration was seen in none among patients or healthy volunteers; whitish discolouration was seen in 15 (50%) patients and 1 (3.33%) healthy volunteer. Only one(3.33%) had normal ;while in healthy volunteers one was affected among 29 .

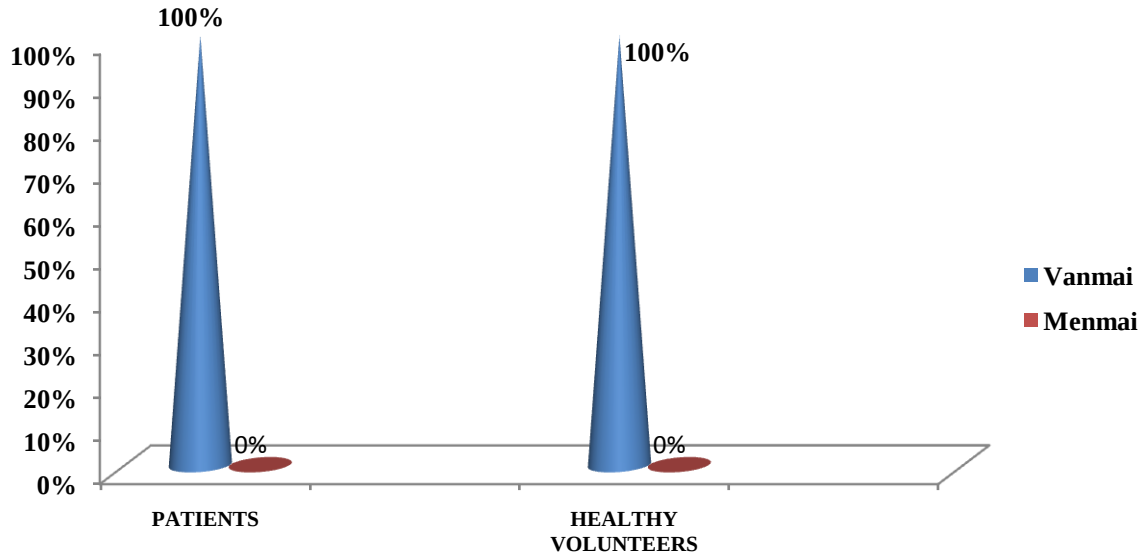
Inference

Majority of patients had manjal (46.66%) and veluppu (50%) discolouration of eyes. Among healthy volunteers majority had normal eye only one (3.33%)had whitish discolouration . Yellowish discolouration of eyes indicates deranged pitha humour in this study also the basic cause for disease is pitha, hence this inference justifies the literature.

Table 25-Naadi

NAADI		Athi nurai neer		Healthy volun teers	
		No.of cases	**Percen tage**	**No.of Cases**	**Percentage**
Naadi nithanam (Pulse Appraisal)	Vanmai	30	100	30	100
	Menmai	0	0	0	0
	Total	**30**	**100**	**100**	**100**
Naadi Panbu (Pulse character)	Thanna dai	30	100	30	100
	Ilaithal	0	0	0	0
	Kuthithal	0	0	0	0
	Thullal	0	0	0	0
	Total	**30**	**100**	**30**	**100**
Naadi Nadai (Pulse play)	Vatha pitham	4	13.33	13	43.33
	Vatha kabam	1	3.33	2	6.66
	Pitha va tham	23	76.66	11	36.66
	Pitha Kapham	2	6.66	0	0
	Kapha vatham	0	0	2	6.66
	Kapha p itham	0	0	2	6.66
	Total	**30**	**100**	**30**	**100**

Figure 22: Naadi nithanam



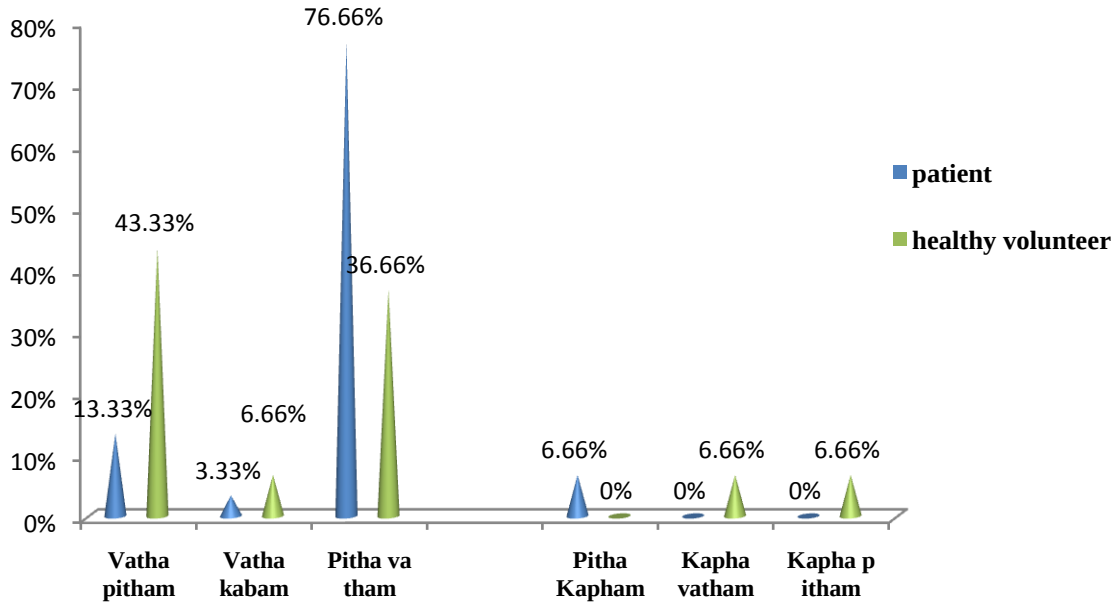
Observation

Out of 30 cases all patients as well as healthy volunteers , when naadi was observed vanmai was present.

Inference

None had menmai pulse appraisal.

Figure 23 – Naadi nadai(Pulse play)



Observation

Out of 30 patients, pulse appraisal and pulse character was normal. In case of pulse play 4 (13.33%) among patients and 13 (43.33%) among healthy volunteers had Vaatha pitham, 1 (3.33%) among patients and 2 (6.66%) among healthy volunteers had Vaatha kabam , 23 (76.66%) among patients and 11 (36.66%) among healthy volunteers had Pitha Vaatham,none among patients and 2 (6.66%) among healthy volunteers had kaba pitham as well as kaba vatham.

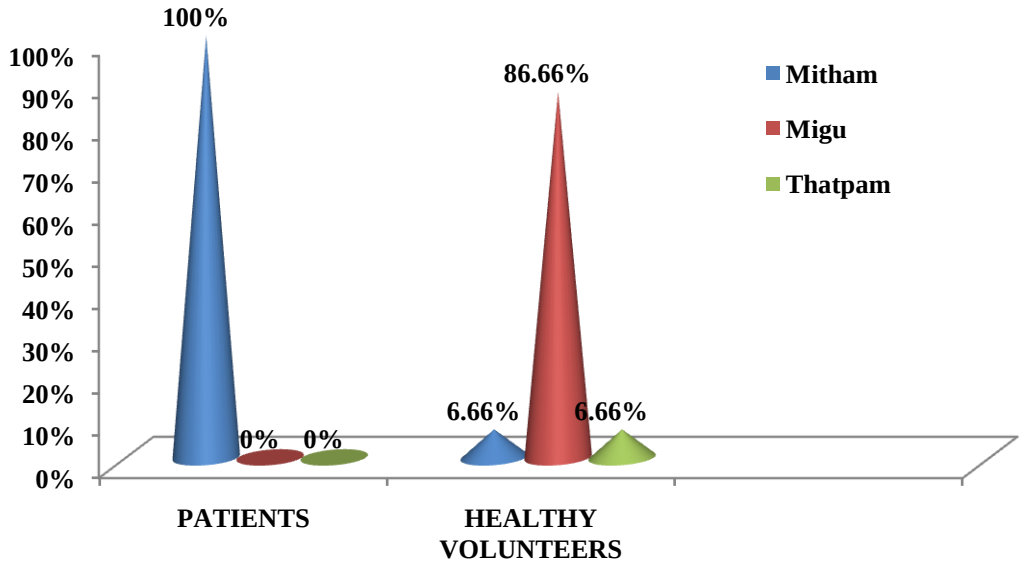
Inference

Majority of patients had Pitha Vaatha naadi(76.66%).Among healthy volunteers Vaatha Pitha naadi seemed to common. Since pitham humour is the driving force of Madhumegham that may be the reason for pitha vatham or vatha pitham naadi in majority of the cases.

Table -26 Sparisam

Mei kuri		Athi Nurai Neer No.of cases	Percentage	No.of Healthy Volunteers	Percentage
Veppam	Mitham	30	100	2	6.66
	Migu	0	0	25	86.66
	Thatpam	0	0	2	6.66
	Total	**30**	**100**	**30**	**100**
Viyarvai	Iyalbu	30	100	26	86.66
	Athigam	0	0	2	6.66
	Total	**30**	**100**	**2**	**6.66**
Thanmai	Thodu vali	8	26.66	0	0
	Udal varatchi	0	0	0	0
	Iyalbu	22	73.33	30	100
	Total	**30**	**100**	**30**	**100**

Figure 24 – Meikkuri - Veppam



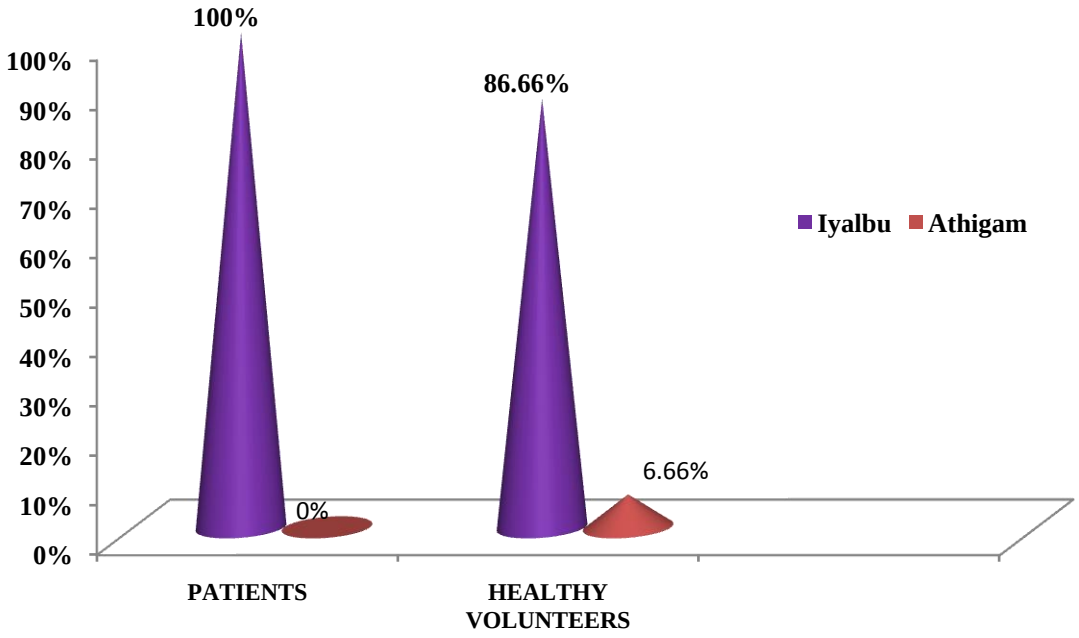
Observation

Out of 30 cases all patients had mitha Veppam, in case of healthy volunteers 25 had migu veppam, 2 had thatpam.

Inference

Meikkuri was not so significant in this study all patients had mithu veppam.

Figure 25 – Meikkuri - Viyarvai



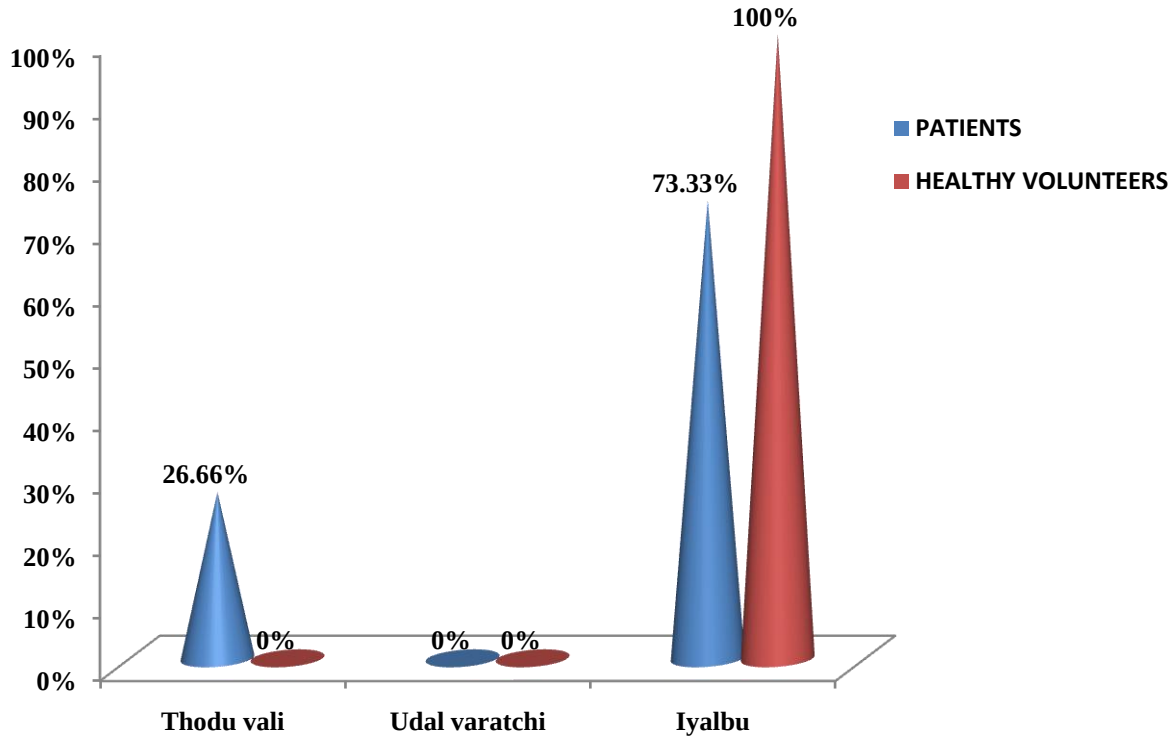
Observation

Out of 30 cases sweating was normal for all patients, excessive in 2(6.66%) and reduced in 2(6.66%) healthy volunteers.

Inference

Majority of patients as well as healthy volunteers had normal sweating, sweating was not a significant factor in this study.

Figure 26- Thanmai



Observation

Out of 30 cases tenderness (thodu vali) was present in 8(26.66%) cases, dryness of skin was not seen in either of patients or healthy volunteers.

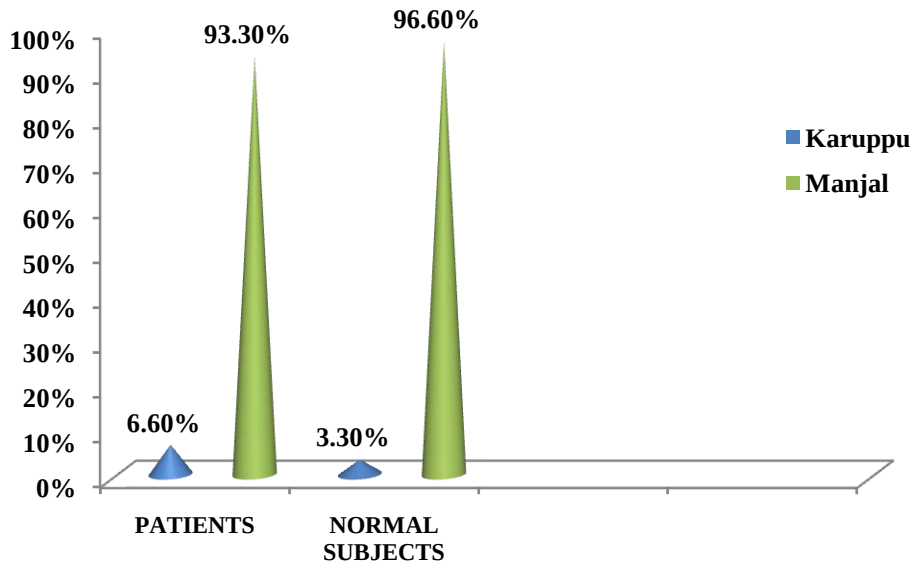
Inference

Thodu vali was seen in only eight cases other factors such as udal varatchi was not seen in any of the patients.

Table 27- Malam

Malam		Athi Nurai Neer No of cases	Percentage	No.of Healthy volunteers	Percentage
Niram	Karuppu	2	6.66	1	3.33
	Manjal	28	93.33	29	96.66
	Total	**30**	**100**	**30**	**100**
Thanmai	Mala Sikkal	3	10	8	26.66
	Sirutthal	0	0	0	0
	Kalichchal	0	0	0	0
	Seetham	0	0	0	0
	Total	**30**	**100**	**30**	**100**

Figure 25- Malam- Niram



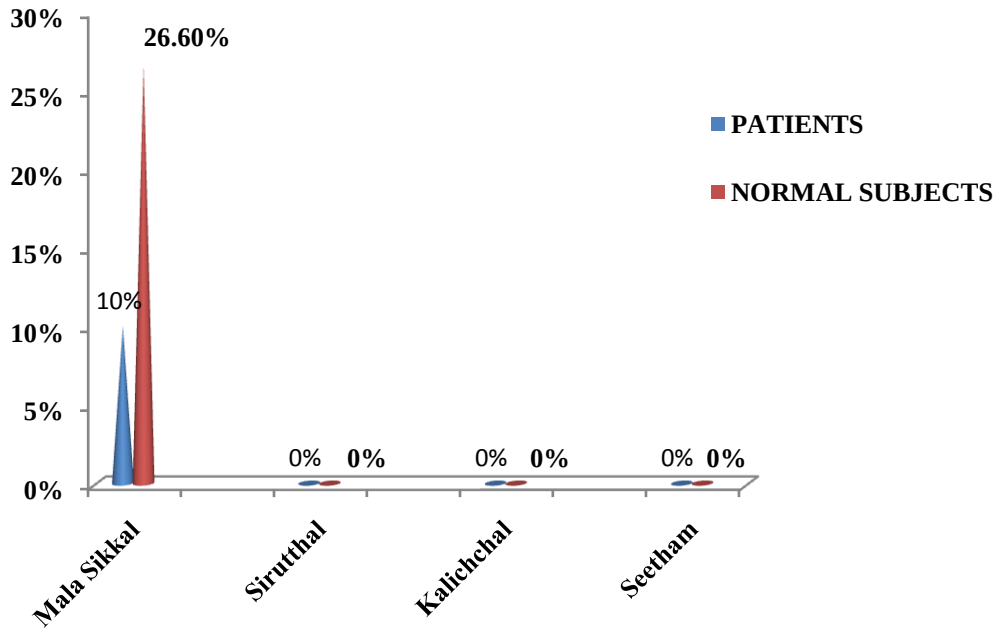
Observation

Out of 30 cases 2 (6.66%) patients,1 (3.33%) healthy volunteer had blackish discolouration of feces, 28 (93.33%) patients and 29 (96.66%) healthy volunteers had yellow coloured feces, constipation was seen in 3 (10%) patients and 8 (26.66%) normal subjects. Poorly formed stools, diarrhea, mucous discharge was absent in all cases.

Inference

Majority of patients as well as healthy volunteers had yellowish coloured feces, only few had blackish discolourated, and constipation was seen only in 3 among patients and 8 among healthy volunteers.

Figure 26- Malam- Thanmai



Observation

Out of 30 cases constipation was seen in 3 (10%) of patients and 8 (26.66%) of normal subjects. Poorly formed stools, diarrhea, mucous discharge was absent in all cases.

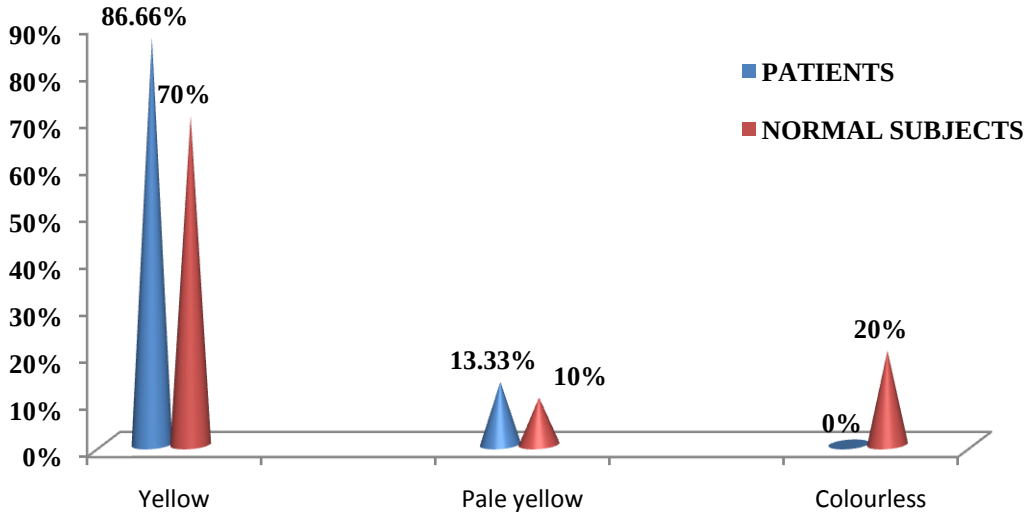
Inference

Since the disease is not associated with gastro intestinal tract there was no complaints of diarrhea, mucous or bloody discharge.

Table 28-Moothiram

Neer kuri		Athi nurai neer		No.of Healthy Volunteer	Percen Tage
		No.of Cases	Percent age		
Neer Thanmai	Neer Manam(Ammonical)	30	100	30	100
	Fruity	0	0	0	0
	Total	**30**	**100**	30	100
Neer Niram	Yellow	26	86.66	21	70
	Pale yellow	4	13.33	3	10
	Colourless	0	0	6	20
	Total	**30**	**100**	**30**	**100**
Nurai	Absent	0	0	30	100
	Present	30	100	0	0
	Total	**30**	**100**	**30**	**100**
Edai	Iyalbu	30	100	30	100
	Total	**30**	**100**	**30**	**100**
Enjal	Iyalbu	20	66.66	30	100
	Athigam	10	33.33	0	0
	Total	**30**	**100**	**30**	**100**

Figure 27 – Colour of urine



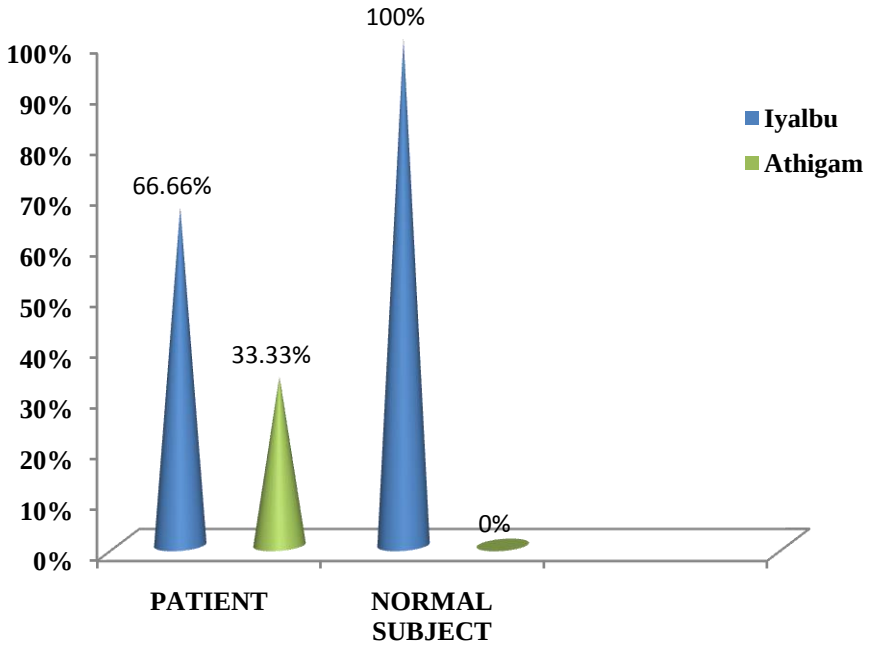
Observation

Out of 30 patients, yellow colour urine was seen in 26(86.66%) patients and 21(70%) healthy volunteers; pale yellow colour was seen in 4(13.33%) patients and 3 (10%) healthy volunteers, and colourless in 6 (20%) healthy volunteers.

Inference

Majority of patients as well as healthy volunteers had yellow coloured urine, which is an indication of deranged pitham.

Figure 28 – Enjal (Deposits)



Observation

Deposits were found in 10 (33.33%) patients, but not in any of the normal subjects.

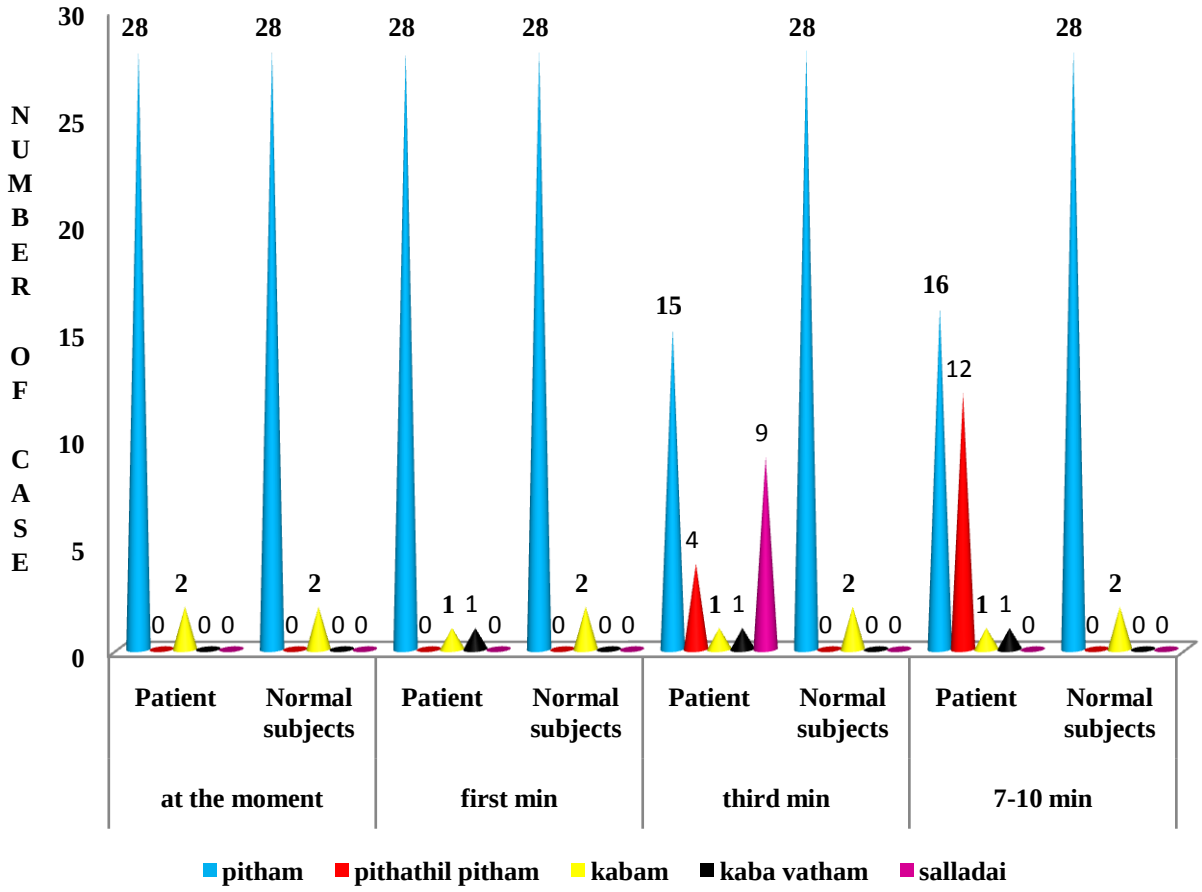
Inference

Urinary tract infection in diabetic condition may be the reason for presence of deposits(pus cells, bacteria) in urine.

Table 29 - Neikkuri

NEIKKURI						
SL NO	AT THE MOMENT	FIRST MINUTE	THIRD MINUTE	TENTH MINUTE	MICRO/ MACRO ALBUMINURIA	ETIOLOGY
1	Kabam	Kabam	Kabam	Kabam	Macro	Diabetic
2	Pitham	Pitham	Pitham	Pitham	Macro	Diabetic
3	Pitham	Pitham	Pitham	Pitham	Micro	Diabetic
4	Pitham	Pitham	Pitham	Pitham	Micro	Diabetic
5	Pitham	Pitham	Pitham	Pitham	Micro	Diabetic
6	Pitham	Pitham	Salladai	Pithathil pitham	Micro	UTI
7	Pitham	Pitham	Pithathil pitham	Pithathil pitham	Macro	Diabetic
8	Pitham	Pitham	Salladai	Pithathil pitham	Macro	Diabetic
9	Pitham	Pitham	Salladai	Pithathil pitham	Macro	Diabetic
10	Kabam	Kaba Vatham	Kaba Vatham	Kaba Vatham	Macro	UTI
11	Pitham	Pitham	Pitham	Pitham	Macro	UTI
12	Pitham	Pitham	Pitham	Pithathil pitham	Micro	Diabetic
13	Pitham	Pitham	Salladai	Pitham	Micro	Diabetic
14	Pitham	Pitham	Pitham	Pitham	Macro	Diabetic
15	Pitham	Pitham	Pitham	Pitham	Micro	Diabetic
16	Pitham	Pitham	Pitham	Pitham	Micro	Diabetic
17	Pitham	Pitham	Salladai	Pithathil pitham	Micro	Diabetic
18	Pitham	Pitham	Salladai	Pithathil pitham	Macro	Diabetic
19	Pitham	Pitham	Pitham	Pitham	Micro	Diabetic
20	Pitham	Pitham	Pitham	Pitham	Macro	Diabetic
21	Pitham	Pitham	Salladai	Pithathil pitham	Micro	Diabetic, UTI, BPH
22	Pitham	Pitham	Pitham	Pitham	Macro	Diabetic
23	Pitham	Pitham	Pitham	Pitham	Micro	Diabetic
24	Pitham	Pitham	Pitham	Salladai	Micro	Diabetic
25	Pitham	Pitham	Pitham	Salladai	Macro	Diabetic
26	Pitham	Pitham	Pithathil pitham	Pithathil pitham	Macro	Diabetic
27	Pitham	pitham	Pithathil pitham	Pithathil pitham	Micro	UTI
28	Pitham	Pitham	Pithathil pitham	Pithathil pitham	Micro	Diabetic
29	Pitham	Pitham	Pitham	Pitham	Micro	Diabetic
30	Pitham	Pitham	Pitham	Pitham	Micro	Diabetic

Figure 28 – Neikkuri



Observation

Out of thirty patients 28(93.33%) had pitham pattern at the moment when the oil was instilled, 2 (6.66%) had kabam pattern. Among 30 healthy healthy volunteers 28 (93.33%) had pitham at the moment and 2 (6.66%) had kabam.

During first minute when the spreading of oil was observed, 28 subjects both from patients and healthy volunteers remained as pitham, but got spread symmetrically; one among the patient who showed kabam at the moment turned to kaba vatham when observed at the first minute.

During the third minute observation among the 28 patients who showed pitham at first moment 15 remained as pitham; 4 turned pithathil pitham 9 turned to salladai. One patient with kabam remained kabam. One with kaba vatham remained kaba vatham. While in case of normal subjects 28 who showed pitham remained as such. 2 with kabam remained as such.

During the 7-10 th minute observation 16 patients showed pitham; 12 showed pithathil pitham; 1 kabam, 1 kabavatham.Among normal subjects 28 showed pitham, 2 showed kabam.

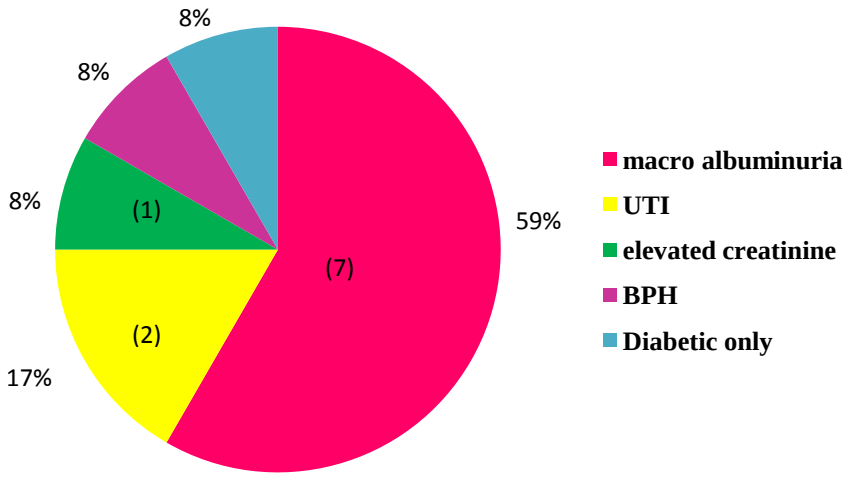
Inference

Pitham was the affected factor in majority of the patients (28) among them twelve showed pithathil pitham which may the indicator of severity of the condition. From this it can be said that since all the patients belonged to mega roga which is a disease due to the derangement of pitham, here the Neikkuri result showed the affected humour.

Table 30-Pithathil pitham Neikkuri pattern

Sl No	PITHATHIL PITHAM	
1	Macroalbuminuria	7
2	UTI	2
3	Elevated creatinine	1
4	BPH	1
5	Diabetic only	1
6	**Total**	**12**

Figure - 29



Observation

Out of 30 patients 12(40%) patients showed Neikkuri as pithathil pitham.Among the twelve 7 (59%) were having macro aluminuria, 2 (17%) had UTI, 1 (8%) had elevated creatinine, 1 (8%) had BPH and one had diabetes only.

Inference

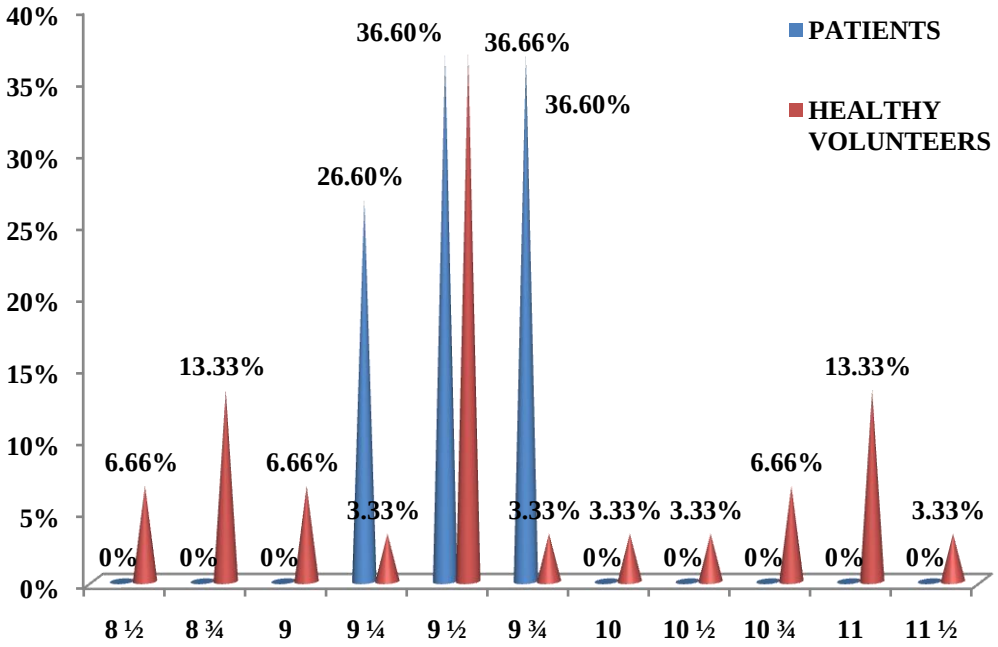
From this it can be said that pithathil pitham in Neikkuri was shown by patients with macroalbuminuria, elevated creatinine and UTI ; these all comes under pitha elevated conditions because renal system it is the dwelling place of pitha humour so diseases related to renal system can be said to be a condition with elevated pitha.

9.16 MANIKADAI NOOL

Table 31- Manikadai nool

Sl No	Manikkadai Nool(Virakadai)	Athi nurai neer		No of healthy volunteers	Percentage
		No of cases	Percentage		
1	8 ½	0	0	2	6.66
2	8 ¾	0	0	4	13.33
3	9	0	0	2	6.66
4	9 ¼	8	26.66	1	3.33
5	9 ½	11	36.66	11	36.66
6	9 ¾	11	36.66	1	3.33
7	10	0	0	1	3.33
8	10 ½	0	0	1	3.33
9	10 ¾	0	0	2	6.66
10	11	0	0	4	13.33
11	11 ½	0	0	1	3.33

Figure 30 – Manikkadai nool



Observation

Out of 30 patients, 8 (26.66%) had manikkadai nool measurement was 9 ¼ finger breadth; 1 (3.33%) among healthy volunteer had 9 ¼ finger breadth. 11 patients (36.66%) had 9 ½ finger breadth. Among healthy volunteers, 11 (36.66%) had also 9 ½. 11 (36.66%) among 30 patients had 9 ¾ finger breadth, and 1(3.33%) among healthy volunteers.

8 ½ finger breadth measurement was present in none of the patients but in 2(6.66%) healthy volunteers, 8 ¾ finger breadth measurement was seen in none of the patients but in 4(13.66%) healthy volunteers, 9 finger breadth measurement was seen in none of the patients but 2 (6.66%) of healthy volunteers, 10 and 10 ½ finger breadth measurement was seen in none among the patients but in 1 (3.33%) among healthy volunteers .10 3/4 finger breadth measurement was seen in 2 (6.66%) of healthy volunteers.11 finger breadth was seen in none of the patients but 4 (13.33%) healthy volunteers.11 ½ finger breadth measurement was seen in none of the patients but in 1 (3.33%) healthy volunteers.

Inference

Majority of patients had 9 ½ finger breadth measurements. It is stated that there is susceptibility for occurrence of mega diseases for 9 ½ finger breadth according to *Agathiyar Soodamani Kaiyiru Soothiram.*

Table 32 LABORATORY INVESTIGATIONS OF PATIENTS WITH ATHI NURAI NEER

Subject No	OP/IP No	TC	DC			Hb	ESR		blood sugar		CHOLESTEROL			TRIGLYCERIDES	BLOOD UREA	SERUM CREATININE
			P	L	Mixed		30 min	60 min	fasting	post prandial	Sr.CHOL	HDL	LDL			
1	H47024	6500	65	35	0	13.8	4	8	124	200	140	48	35	140	19	0.9
2	H47125	6200	68	30	2	12.3	4	10	121	140	204	37	101	73	20	0.8
3	H79792	9500	69	28	3	13.8	12	24	152	178	216	64	118	133	18	0.9
4	H16460	4800	60	35	5	13.3	12	24	145	244	160	45	90	168	16	1
5	H67384	7200	69	30	1	11.9	4	10	118	164	140	52	80	43	16	0.8
6	H98173	12100	72	25	3	11.8	4	18	101	115	120	65	88	110	16	0.9
7	J88522	6500	69	30	1	12	5	10	220	290	125	56	90	159	22	1.9
8	J87464	6900	70	28	2	12.5	5	10	165	221	166	65	98	145	16	1
9	J14777	14800	87	11	2	13	10	22	217	336	190	57	114	102	20	1
10	J54423	8800	54	44	2	11.1	10	20	102	114	130	55	112	150	12	0.5
11	J39014	5000	66	30	4	12.5	12	24	93	100	175	68	90	86	18	0.7
12	J93623	6300	54	41	5	13.5	2	6	190	268	129	32	77	102	26	1
13	J88821	6400	70	28	2	14.9	8	10	72	152	287	51	171	103	40	2
14	j91332	6500	70	28	2	12.3	10	14	194	240	150	56	120	110	29	0.7
15	J94535	7800	73	24	3	12.5	2	6	177	250	120	56	110	167	12	0.6

Table 33 LABORATORY INVESTIGATIONS OF PATIENTS WITH ATHI NURAI NEER

Subject No	OP/IP No	TC	DC			Hb	ESR		blood sugar		CHOLESTEROL			TRIGLYCERIDES	BLOOD UREA	SERUM CREATININE
			P	L	Mixed		30 min	60 min	fasting	post prandial	Sr.CHOL	HDL	LDL			
16	J98667	6200	68	30	4	11.5	4	6	123	230	150	55	60	151	30	1.2
17	J86420	10200	69	30	60	13	4	6	120	150	190	52	108	120	16	0.5
18	I05859	6400	76	20	4	12.5	4	6	120	140	110	58	100	101	32	1.2
19	J93674	6500	75	21	4	14.2	4	6	110	160	179	48	103	141	30	1.2
20	I16591	10300	77	21	2	12.9	16	34	110	156	120	55	65	145	16	0.7
21	J1392	6400	68	30	2	13.5	10	20	129	160	250	37	99	130	31	1.2
22	G13110	6800	73	24	3	14.1	12	24	109	133	150	35	89	130	18	1.1
23	H24458	5700	67	25	2	12.4	12	20	89	110	250	40	78	150	24	0.7
24	H91510	6000	70	26	4	12.5	4	6	180	269	150	45	159	150	16	0.8
25	J88034	8800	54	44	2	12	60	100	165	202	120	45	140	150	19	1
26	J88451	9900	74	22	4	11	20	44	120	200	90	40	145	150	18	1
27	J85462	6600	61	33	6	12.8	10	22	95	100	58	124	126	150	43.5	2.4
28	J76242	8500	77	20	2	10	22	46	90	110	42	98	112	120	18	0.8
29	I63594	7000	65	30	5	14.2	11	20	120	150	32	124	140	150	16	0.9
30	K11780	7800	72	22	6	13.5	2	4	110	156	100	123	145	130	17	0.9

TABLE 34 LABORATORY INVESTIGATIONS OF PATIENTS

Subject No	OP/IP No	Urine			24 Hour Urine Protein	Spot Miroalbumin	tenth minute (7-10Minutes)	leukocyte	nitrate	pH	spedific gravity	blood	ketone
		albumin	sugar	deposits									
1	H47024	(++)	Nil	1-2hpf	4649.8		Muthu	(-)	(-)	Acidic	1.025	(-)	(-)
2	H47125	(+)	Nil	Nil		368	Coin	(-)	(-)	Acidic	1.025	(-)	(-)
3	H79792	(+)	Nil	Nil		276	Round	(-)	(-)	Acidic	1.025	(-)	(-)
4	H16460	(+)	Nil	1-2hpf	154		Coin	(-)	(-)	Acidic	1.025	(-)	(-)
5	H67384	(+)	Nil	2-4 hpf		61	Coin	(-)	(-)	Acidic	1.025	(-)	(-)
6	H98173		Nil	8-10 hpf		22	Pithathil pitham	(-)	(-)	Acidic	1.025	(-)	(-)
7	J88522	(+++)	(+++)	3-5 hpf		1861	Pithathil pitham	(-)	(-)	Acidic	1.025	(-)	(-)
8	J87464	(+++)	(+)	1-2hpf			Pithathil pitham	(-)	(-)	Acidic	1.025	(-)	(-)
9	J14777	(++)	(++)	8-10 hpf	3105		Pithathil pitham	(-)	(-)	Acidic	1.025	(-)	(-)
10	J54423	(+)	Nil	plenty of bacteria		29.67	Kaphavatham	(-)	(-)	Acidic	1.025	(-)	(-)
11	J39014	(++)	Nil	plenty of bacteria		266.38	Round	(-)	(-)	Acidic	1.025	(-)	(-)
12	J93623	(+)	Nil	1-2hpf		30	Round	(-)	(-)	Acidic	1.025	(-)	(-)
13	J88821	(+)	Nil	1-2hpf		4130.7	Pithathil pitham	(-)	(-)	Acidic	1.025	(-)	(-)
14	j91332	(+)	Nil	1-2hpf		125	Round	(-)	(-)	Acidic	1.025	(-)	(-)
15	J94535	(+)	(+)	1-2hpf		213	Round	(-)	(-)	Acidic	1.025	(-)	(-)

Subject No	OP/IP No	Urine			24 Hour Urine Protein	Spot Miroalbumin	tenth minute (7-10Minutes)	leukocyte	nitrate	pH	spedific gravity	blood	ketone
		albumin	sugar	deposits									
16	J98667	(+)	(+)	1-2hpf		19	Coin	(-)	(-)	Acidic	1.025	(-)	(-)
17	J86420	(+)	(+)	1-2hpf	105		Pithathil pitham	(-)	(-)	Acidic	1.025	(-)	(-)
18	I05859	(++)	Nil	1-2hpf			Pithathil pitham	(-)	(-)	Acidic	1.025	(-)	(-)
19	J93674	(++)	Nil	1-2hpf			Round	(-)	(-)	Acidic	1.025	(-)	(-)
20	I16591	(++)	Nil	bacteria loaded		886	Round	(-)	(-)	Acidic	1.025	(-)	(-)
21	J1392	(+)	Nil	8-10 pus cells	50		Pithathil pitham	(-)	(-)	Acidic	1.025	(-)	(-)
22	G13110	(+)	Nil	1-2hpf	109.2		Round	(-)	(-)	Acidic	1.025	(-)	(-)
23	H24458	(+)	Nil	1-2hpf		20.4	Round	(-)	(-)	Acidic	1.025	(-)	(-)
24	H91510	(++)	(++)	1-2hpf		3810.8	Pithathil pitham	(-)	(-)	Acidic	1.025	(-)	(-)
25	J88034	(++)	Nil	8-10 pus cells	3441		Pithathil pitham	(-)	(-)	Acidic	1.025	(-)	(-)
26	J88451	(+++)	(+)	bacteria loaded			Coin	(-)	(-)	Acidic	1.025	(-)	(-)
27	J85462	(+)	nil	plenty of pus cells	29.7		pithathilpitha	(-)	(-)	Acidic	1.025	(-)	(-)
28	J76242	(++)	nil	1-2hpf	227		pithathilpitha	(-)	(-)	Acidic	1.025	(-)	(-)
29	I63594	(+)	nil	motile bacilli	38.2		Round	(-)	(-)	Acidic	1.025	(-)	(-)
30	K11780	(+)	nil	1-2hpf	271		Round	(-)	(-)	Acidic	1.025	(-)	(-)

TABLE 35 LAB INVESTIGATIONS OF HEALTHY VOLUNTEERS

Subject No	OP/IP No	TC	DC			Hb	ESR		blood sugar		CHOLESTEROL		TRIGLYCERIDES	BLOOD UREA	SERUM CREATININE
			P	L	Mixed		30 min	60 min	fasting	post prandial	Serum .Cholesterol	HDL			
1	**J86520**	8700	60	37	3	14.1	30	60	98	164	55	134	132	14	0.8
2	**J41187**	9000	67	30	3	11.3	3	9	87	139	65	86	76	15	0.9
3	**J85023**	9200	59	39	2	12.3	6	8	96	132	40	98	142	39	0.8
4	**J77239**	8600	59	39	2	16.3	2	4	80	110	43	100	85	37	1.1
5	**J90431**	8700	70	25	5	12.4	8	16	84	115	62	79	74	16	0.7
6	**I38610**	9700	56	40	5	12	8	10	98	115	42	99	112	20	1.1
7	**J97061**	14400	71	27	2	11.4	20	42	118	124	37	85	156	27	0.6
8	**J83647**	6000	70	23	7	15.4	2	4	105	115	52	139	263	20	1.1
9	**J99847**	6300	64	34	2	15.8	12	24	96	132	51	95	127	20	1.1
10	**J99614**	7600	58	37	5	10.9	50	100	92	121	29	75	113	8	0.9
11	**K02746**	8400	51	44	5	14.5	2	6	82	133	42	59	112	20	1.1
12	**K01560**	5400	71	24	5	10.9	8	16	88	76	49	71	45	9	0.7
13	**K07065**	9900	52	46	2	15.7	4	10	95	132	47	157	284	35	0.8
14	**J79076**	5500	57	41	2	12.1	22	44	98	119	54	128	290	21	1
15	**K05103**	7900	68	28	4	14	4	8	100	142	49	96	129	19	0.8
16	**K06023**	8500	60	37	3	12	12	16	110	154	40	102	140	17	1.5
17	**K03015**	8800	63	34	3	10.3	10	22	109	209	67	106	93	19	1
18	**K09497**	11600	59	39	2	11.9	4	10	102	124	39	137	235	16	1
19	**K06459**	6800	56	40	4	11.5	26	54	94	104	56	128	236	18	0.8
20	**K09129**	8600	66	32	2	14.1	12	26	113	121	48	152	154	17	1.1
21	**J81698**	3200	55	42	3	11	20	42	76	110	49	88	136	13	0.7
22	**K05083**	7500	67	30	3	11.3	8	12	79	110	43	28	142	18	1
23	**K08141**	8300	71	19	10	9.7	30	62	88	110	40	132	150	15	1
24	**K00926**	6000	59	34	7	14.4	12	19	72	125	36	72	134	14	1.1
25	**J85776**	5800	60	38	2	13.3	4	8	88	160	42	100	108	16	0.7
26	**J97395**	6100	73	20	7	13.6	20	10	96	111	42	65	89	16	1.2
27	**K18655**	5400	62	26	12	11.1	15	30	91.1	86.2	42.6	111.1	86.2	18.4	0.63
28	**K05384**	7700	74	24	2	10.3	4	8	83	122	40	86	115	16	0.9
29	**K12774**	6600	59	38	3	13.1	50	102	50	102	133	152	137	11	0.8
30	**K21961**	7000	55	40	5	13.5	2	4	122	152	35	110	129	35	0.8

Table 36 LABORATORY INVESTIGATIONS OF HEALTHY VOLUNTEERS

Subject No	OP/IP No	Urine				
		albumin	sugar	deposits	pH	Specific gravity
1	**J86520**	Nil	Nil	2-4 hpf	Acidic	1.025
2	**J41187**	Nil	Nil	2-4hpf	Acidic	1.005
3	**J85023**	Nil	Nil	2-4hpf	Acidic	1.005
4	**J77239**	Nil	Nil	2-4hpf	Acidic	1.025
5	**J90431**	Nil	Nil	1-2hpf	Acidic	1.025
6	**I38610**	Nil	Nil	4-8hpf	Acidic	1.025
7	**J97061**	Nil	Nil	3-5 hpf	Acidic	1.025
8	**J83647**	Nil	Nil	3-4hpf	Acidic	1.005
9	**J99847**	Nil	Nil	1-2hpf	Acidic	1.005
10	**J99614**	Nil	Nil	2-4hpf	Acidic	1.025
11	**K02746**	Nil	Nil	2-4hpf	Acidic	1.025
12	**K01560**	Nil	Nil	2-4hpf	Acidic	1.025
13	**K07065**	Nil	Nil	1-2hpf	Acidic	1.025
14	**J79076**	Nil	Nil	2-3hpf	Acidic	1.025
15	**K05103**	Nil	Nil	2-4hpf	Acidic	1.025
16	**K06023**	Nil	Nil	2-4hpf	Acidic	1.025
17	**K03015**	Nil	Nil	2-4hpf	Acidic	1.025
18	**K09497**	Nil	Nil	1-2hpf	Acidic	1.025
19	**K06459**	Nil	Nil	2-4hpf	Acidic	1.025
20	**K09129**	Nil	Nil	2-4hpf	Acidic	1.025
21	**J81698**	Nil	Nil	2-4hpf	Acidic	1.025
22	**K05083**	Nil	Nil	1-2hpf	Acidic	1.025
23	**K08141**	Nil	Nil	2-3hpf	Acidic	1.025
24	**K00926**	Nil	Nil	2-4hpf	Acidic	1.025
25	**J85776**	Nil	Nil	2-4hpf	Acidic	1.025
26	**J97395**	Nil	Nil	2-4hpf	Acidic	1.025
27	**K18655**	Nil	Nil	1-2hpf	Acidic	1.025
28	**K05384**	Nil	Nil	2-4hpf	Acidic	1.025
29	**K12774**	Nil	Nil	2-4hpf	Acidic	1.025
30	**K21961**	Nil	Nil	2-4hpf	Acidic	1.025

Athi Nurai Neer is a condition in which urine appears frothy due to derangement of *Tridoshas* as per Siddha concept. Frothy urine is mostly presented in proteinuria conditions. So patients subjected to this study were having proteinuria as well as showed froth in urine. Protienuria indicates degree of renal damage caused by different diseases. Since majority of patients subjected to study were diabetics with albuminuria it can be taken as diabetic nephropathy after repeated investigations. Protienuria can be assessed by simple dipstick methods which is not accurate moreover there are chances of false positive and false negative results, as per researches 24 hour urine protein is considered to be comparatively accurate.

Validity of the research, if it is done in a proper way it is cost effective and takes less time to do. One of the main benefit is if a diagnosis is done in Siddha way, it will be easy to treat the disease on *tridosha* basis and will be effective than empirically administering medicines for mere symptoms. Even though by Naadi we can find out the disease, or the affected *dosha* it may not be concordant when compared with Neerkkuri and Neikkuri findings as pulse reading is a subjective assessment and that there could be inter observer variations.

In this study the author had screened 70 patients with complaints of Athi nurai neer (Frothy urine) and albuminuria from the outpatient Department of National Institute of Siddha. Among those 70 cases, 30 cases were enrolled in the study and observed for symptoms and signs. Out of 30 cases 28 patients were diabetic and out of this 28, two were having Urinary Tract Infection along with Diabetes, and two were having Systemic hypertension along with Diabetes. Two patients among total subjects with proteinuria had Urinary Tract Infection without Diabetes. Anyhow either Diabetes or Urinary Tract Infection both was the possible causes for proteinuria. As per *Siddha* system both are classified or falls under *Mega Rogam*, which is due to derangement of *Pitha* humour.

Distribution of cases by Age group

Among 30 cases 24 cases that is 80% belonged to the category of age group 34-66 years, 0nly 20% belonged to 66-100 years category. None belonged to 0-33 category. 80 % of the cases belonged to the age category 34-66 years. Middle age group patients reported more in NIS for the study condition. And no reporting was recorded in elderly age group.

Distribution of cases by Sex

60 % of selected cases were males, as per studies prevalence of diabetes is equal in men as well as women, but in this hospital based study since the sample size was less it showed more incidence in males when compared to females.

Distribution of cases by Food habits

Most of the cases were being non-vegetarian. Non-vegetarian diet which is considered as thamo gunam food seem to alter the normal constitution of a person and cause disease.

Distribution of cases by Socio- Economic status

Majority of cases belong to middle income group. As per WHO report of 2016, an estimated 347 million people in the world had diabetes and the prevalence is growing particularly in low- and middle income countries; as per this study all the cases came under socio economic status of either low income or middle income group

Distribution of cases by Yaakai Ilakkanam

Majority of cases were of *Vatha Pitha udalinar* (temperament). Patients with Vatha pitha temperament subjects would generally be lean in nature, darker complexion, majority of cases did not have these features since the sample size was very limited it was not able to substantiate the literature **.**

Distribution of cases by Noi utra kaalam

Majority of cases in the study got affected with the disease during *Munpani kaalam* (*Margazhi-Thai*), *Munpani* kaalam has got no relation with the disease according to *siddha* literature.

Distribution of cases by Noi utra nilam

Majority of cases hailed from *Neithal Nilam*. No specific inference is made.

Distribution of cases by Gnanenthiriyam

Only Mei and Kan were affected among Gnanenthiriyas in the patients with complaints of Athi nurai neer. Out of 30 cases 23 subjects were having normal Gnanenthiriyangal. So as per this study gnanenthiriyam did not show affections.

Distribution of cases by Kanmenthiriyam

Kai, Kaal and *Vaai* were affected in *Athinurai neer* cases. *Kaal* was affected in many of the cases it might be due to diabetic neuropathy.

Distribution of cases by Udal thathukkal

Saaram, chenneer, oon, kozhuppu was decreased in majority of cases, among *udal kattukkaal, Saaram* was increased in only one case. *Enbu , Moolai, Sukkilam*/ *Suronitham* was not affected in any of the cases. Majority of cases subjected to study were diabetic, as per *siddha* literatures *udal thathukkal* will get affected one by one in *Madhumegham*; here *saaram, chenneer, oon, kozhuppu* were affected in majority of cases justifying the literature.

Distribution of cases by uyir thathukkal – vatham

Viyanan, Devadathan Samanan were affected in majority of cases, *Viyanan* helps in movement hence due to its derangement, movement restriction was seen any many cases. *Devadathan* produces fatigue in many cases fatigue was present. *Abanan* was affected in five , which manifested as constipation. Other vaayus was not affected in any of the cases. hence . *Samanan* neutralizes other four vayus since other vayus are affected *samanan* was also affected in majority of cases.

Distribution of cases by Uyir thathukkal Azhal

Aalosakam, Saathagam were the affected components of Pitham, any diseased one will be having difficulty in doing desired activities, this was shown by the affected subjects also which is a feature of *saathagam*.

Distribution of cases by Uyir thathukkal Iyyam

Avalambagam and *santhigam* were the only affected components of *kabam*.

Distribution of cases by- Colour of tongue

There was no significance in colour observation of tongue because blackish discolouration and whitish discolouration was shown more by healthy volunteers when compared to patient. Yellowish discolouration of tongue was shown in a higher percentage 80% in patients and 57.57% in healthy volunteers, this may be an indication of deranged *pitham*; but this doesn't seems to be much significant affections.

Distribution of cases by– Taste in tongue

Majority of normal subjects as well patients had normal taste sensation. Even though sweet taste sensation of tongue might be a feature of diabetic patients this was not much observed in patients subjected to this hospital based study.

Distribution of cases by - Salivation

Salivation was not a significant observation because one on each that is patients and healthy volunteers had hypersalivation.

Distribution of cases by Niram, Mozhi and Vizhi

Complexion

Majority of patients as well as healthy volunteers had yellowish complexion, colour does not show much significant affections in this hospital based study.

Mozhi

Majority of patients as well as healthy volunteers had sama oli so mozhi did not show much significant affections in this hospital based study.

Distribution of cases by Vizhi

Majority of patients had manjal (46.66%) and veluppu (50%) discolouration of eyes. Among healthy volunteers majority had normal eye only one (3.33%)had whitish discolouration . Yellowish discolouration of eyes indicates deranged pitha humour in this study also the basic cause for disease is pitha, hence this inference justifies the literature.

Distribution of cases by-Naadi

Naadi nadai (Pulse play)

Majority of patients had Pitha Vatha naadi (76.66%).Among healthy volunteers Vatha Pitha naadi was common. Since pitham humour is the driving force of *Madhumegham* that may be the reason for pitha vatham or vatha pitham naadi in majority of the cases. As per *Sathaka Naadi* it is stated that pitha naadi might be felt in Piramegam, but sole *pitha naadi* was not found in any of the cases.

Distribution of cases by Sparisam

Meikkuri - Veppam

Meikkuri was not so significant in this study since this was a hospital based study

Meikkuri - Viyarvai

Majority of patients as well as healthy volunteers had normal sweating, sweating was not a significant factor in this study.

Distribution of cases by - Thanmai

Thodu vali was seen in only eight cases other factors such as udal varatchi was not seen in any of the patients.

Distribution of cases by- Malam

Malam- Niram

Majority of patients as well as healthy volunteers had yellowish coloured feces, only few had blackish discolourated feces.Constipation was seen only in 3 among patients and 8 among healthy volunteers.

Distribution of cases by Malam- Thanmai

Since the disease is not associated with gastro intestinal tract there was no complaints of diarrhoea, mucous or bloody discharge.

Distribution of cases by-Moothiram

Colour of urine

Majority of patients as well as healthy volunteers had yellow coloured urine, which is an indication of deranged pitha.

Deposits present in urine

Urinary tract infection in diabetic condition is common so that may be the reason for presence of deposits(pus cells, bacteria) in urine..

Neikkuri

According to siddha literature *Pitham* is the driving force for mega diseases *"Thodar pitha vinthai alathu megam varathu*". In this study Neikkuri was observed for all patients at the moment when oil was instilled, first, third and upto tenth minute .Pitham was the affected factor in majority of patients (28) among them twelve showed *pithathil pitham* which might be the indicator of severity of the condition. All the patients were either diabetic or had urinary tract infection, all these comes under elevated pitha conditions, because renal system is the dwelling place of pitha humour so diseases related to renal system could be said to be a condition with elevated pitham.

Pithathil pitham Neikkuri pattern

From this it can be said that pithathil pitham in Neikkuri was seen patients with macroalbuminuria, elevated creatinine and UTI ; these all come under elevated pitha conditions , because renal system is the dwelling place of pitha humour so diseases related to renal system could be said to be a condition with elevated pitha.

Distribution of cases by - Manikadai nool

Majority of patients had 9 ½ finger breadth measurements. It is stated that there is susceptibility for occurrence of mega diseases for 9 ½ finger breadth according to *Agathiyar Soodamani Kaiyiru Soothiram.*

Athi Nurai Neer is a condition described by our Sages, to find out major damage of renal system by means of mere examination of urine. By *Neerkuri, Neikkuri* examination it showed that the main affected uyir thathu is *pitham,* by finding out this, treatment can be properly done. In our system of medicine it is very essential to find out the root cause of the disease then only we will be able to treat the condition well.

Other diagnostic parameters such as *Naadi*, *Sparisam*, *Naa*, *Niram*, *Mozhi*, *Vizhi* showed some significance but was not so marked according to this hospital based study. This study can be made more relevant by increasing the sample size of both Macro Albuminuria and Micro Albuminuria subjects, because in this study patients with Macro Albuminuria showed a different pattern that is ***pithathil pitham*** which was not observed in normal subjects. For finding out the degree of renal damage, in modern system of medicine they use dip stick method, spot microalbumin test which is not accurate moreover there are chances of false positive and false negative results, as per reseaerches 24 hour urine protein test is considered to be comparatively accurate; but take a whole day and is not so easy for the patient to collect it in a proper way and this test is expensive too. If this Neikkuri procedure is well developed it will be very cost effective. Not only because of its cost effectiveness it shows the deranged humour, by identifying the deranged humour treatment will be effective.

But the procedure of Neikkuri needs more standardization in the case of factors like time period in which the sample should be collected, time up to which the spreading of oil drop should be observed . By this study the author wishes to conclude that the driving force for causing Athi Nurai Neer of the selected subjects were *pitham* , because majority of cases were diabetic(deranged humour in diabetes is said to be pitham) and the severity of pitham derangement was observed in Neikkuri as ***pithathil pitham***. If the Neikkuri procedure is more standardized the treatment will become more effective.

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CASE I

Neikkuri - Day 1

At the moment

I Minute

III Minute

X Minute

Neerkkuri

INTERPRETATION:

In this case kabam was shown when Neikkuri was observed upto ten minutes, even though the patient was a known case of diabetes, pitham was not shown, this may be the influence of diet taken by this individual.

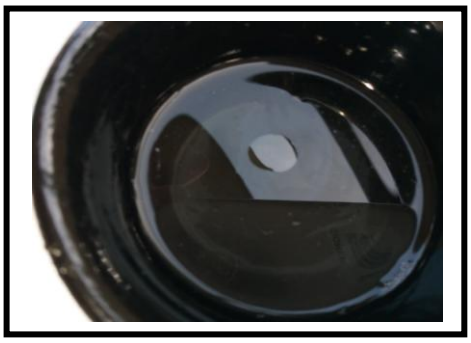
CASE I

Neikkuri Day 2

At the moment

I Minute

III Minute



X Minute

Neerkkuri

INTERPRETATION:

In this case kabam was shown when Neikkuri was observed upto ten minutes, even on the second day so it cannot be merely due to diet, it may be due to the derangement of kabam and its types as stated in literature.

CASE I

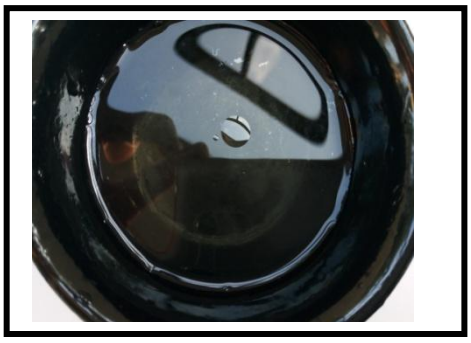
NeikkuriDay 3

At the moment

I Minute

III Minute

X Minute



Neerkkuri

INTERPRETATION:

In this case kabam was shown when Neikkuri was observed upto ten minutes, even on the third day so it cannot be merely due to diet, it may be due to the derangement of kabam and its types as stated in literature.

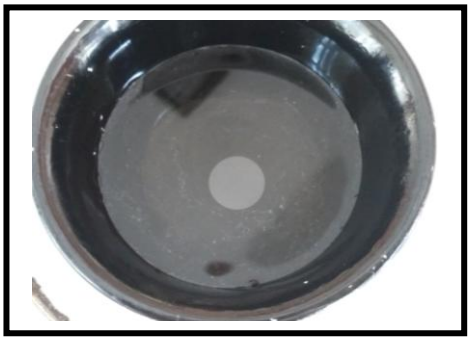
CASE II

Neikkuri -Day 1

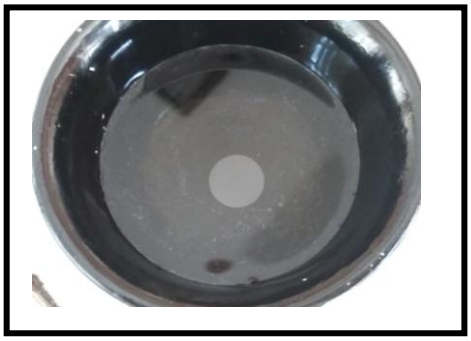
At the moment

I Minute

III Minute



X Minute



Neerkkuri

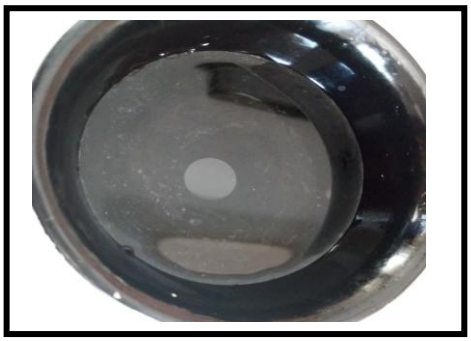
INTERPRETATION:

Patient is a known case of diabetes with macro Albuminuria but the value is 368 mg/ dl, Neikkuri showed pitham which indicates the derangement of the primary causative factor for megham that is pitham.

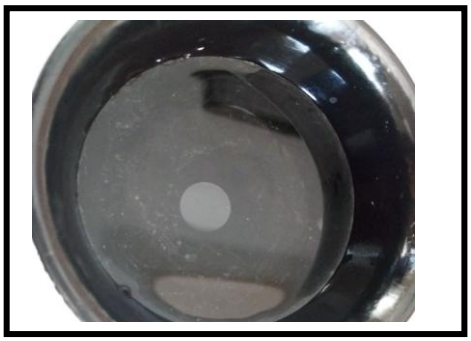
CASE II

Neikkuri Day 2

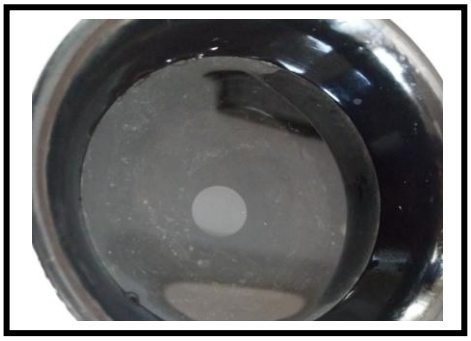
At the moment



I Minute



III Minute



X Minute

Neerkkuri

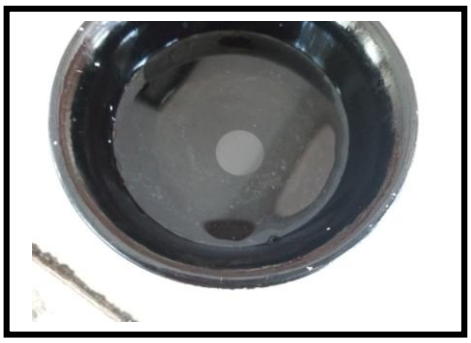
INTERPRETATION:

Even on the second day Neikkuri showed pitham which indicates the derangement of the primary causative factor for megham that is pitham.

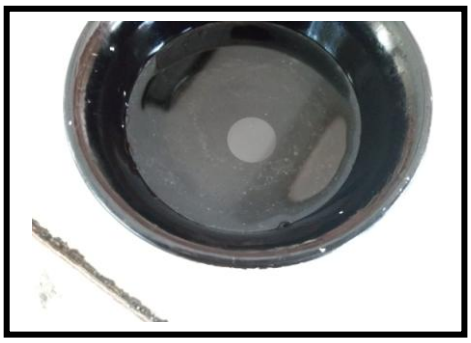
CASE II

NeikkuriDay 3

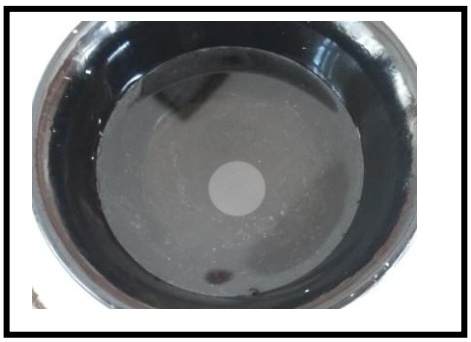
At the moment



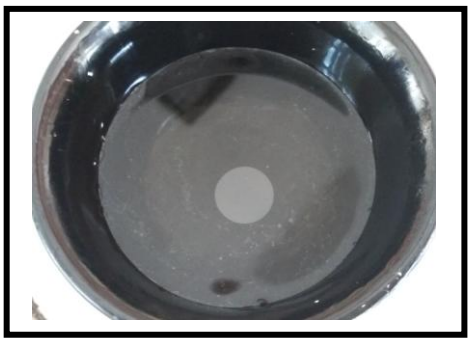
I Minute



III Minute



X Minute



Neerkkuri

INTERPRETATION:

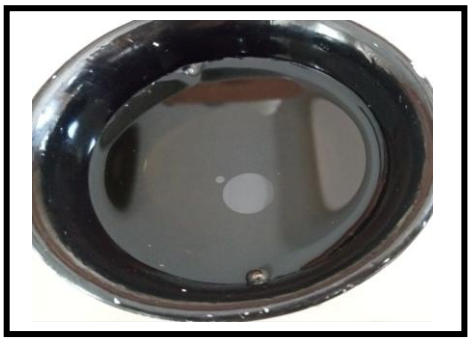
Even on the third day Neikkuri showed pitham which assures the derangement of the primary causative factor for megham that is pitham.

CASE III

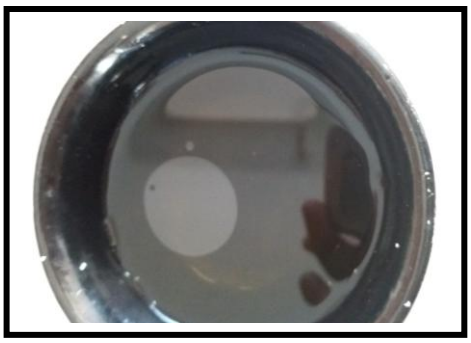
Neikkuri Day 1

At the moment

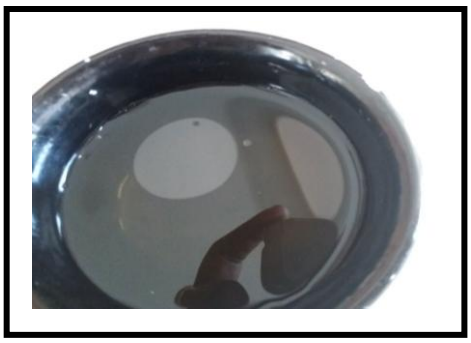
I Minute



III Minute



X Minute



Neerkkuri

INTERPRETATION:

This patient was a known case of type 2 diabetes mellitus with Albuminuria (micro Albuminuria) , there was n urinary tract infection or any associated pitha disease. Patient showed pitham in Neikkuri.

CASE III

Neikkuri Day 2

At the moment

I Minute

III Minute

X Minute

Neerkkuri

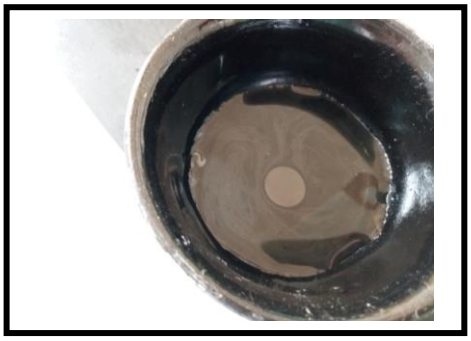
INTERPRETATION:

This patient was a known case of type 2 diabetes mellitus with Albuminuria (micro Albuminuria) , there was n urinary tract infection or any associated pitha disease. Patient showed pitham in Neikkuri on second day also.

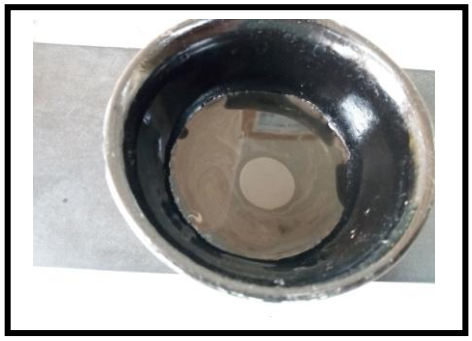
CASE III

Neikkuri Day 3

At the moment

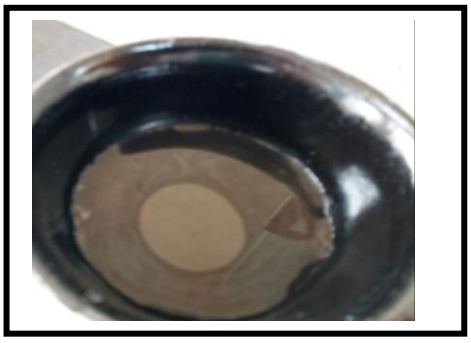


I Minute



III Minute

X Minute



Neerkkuri

INTERPRETATION:

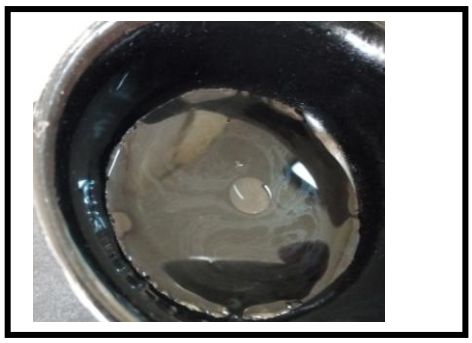
This patient was a known case of type 2 diabetes mellitus with Albuminuria (micro Albuminuria) , there was n urinary tract infection or any associated pitha disease. Patient showed pitham in Neikkuri on third day observation also.

CASE IV

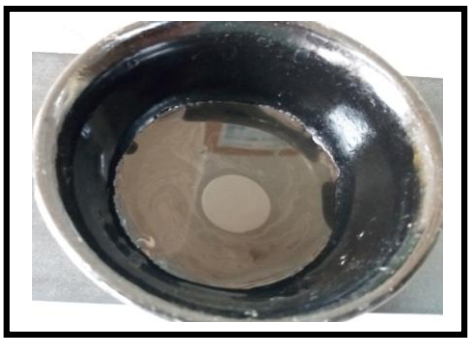
Neikkuri Day 1

At the moment

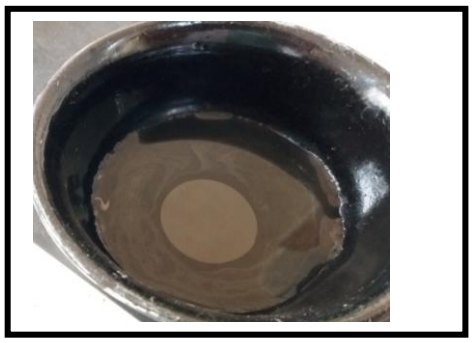
I Minute



III Minute



X Minute



Neerkkuri

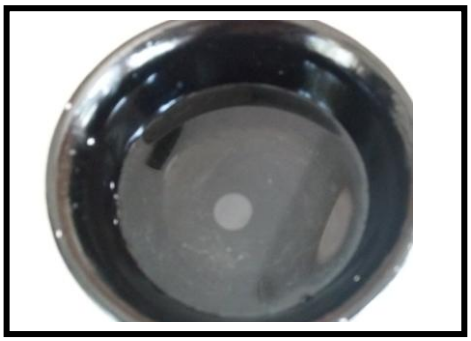
INTERPRETATION:

This patient was a known case of type 2 diabetes mellitus with Albuminuria (micro Albuminuria) , there was n urinary tract infection or any associated pitha disease. Patient showed pitham in Neikkuri on first day observation.

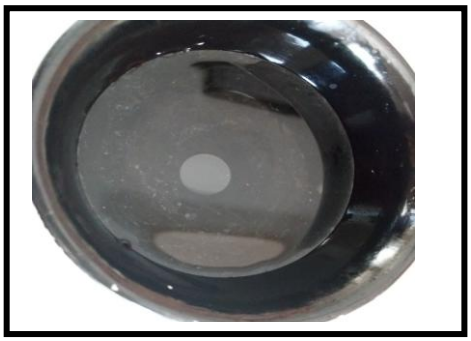
CASE IV

Neikkuri Day 2

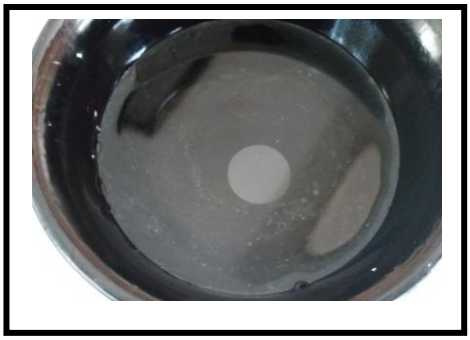
At the moment



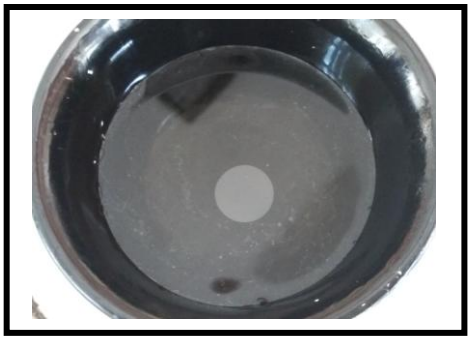
I Minute



III Minute



X Minute



Neerkkuri

INTERPRETATION:

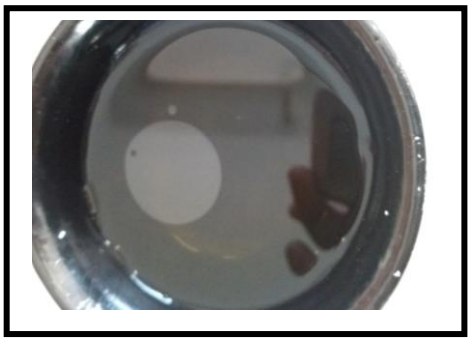
This patient was a known case of type 2 diabetes mellitus with Albuminuria (micro Albuminuria) , there was n urinary tract infection or any associated pitha disease. Patient showed pitham in Neikkuri on day two observation

CASE IV

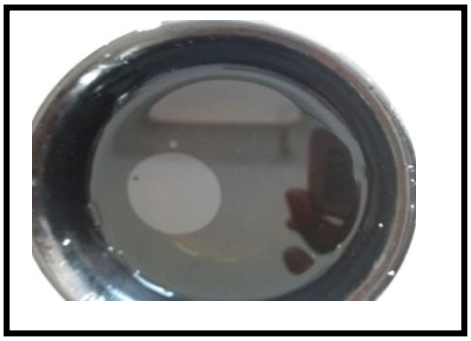
Neikkuri Day 3

At the moment

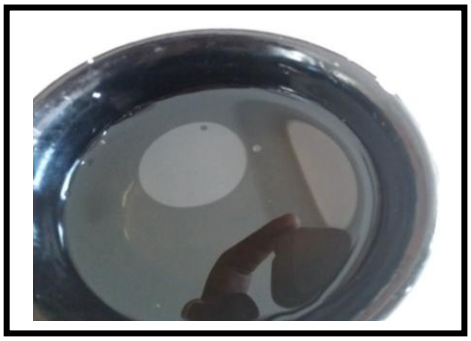
I Minute



III Minute



X Minute



Neerkkuri

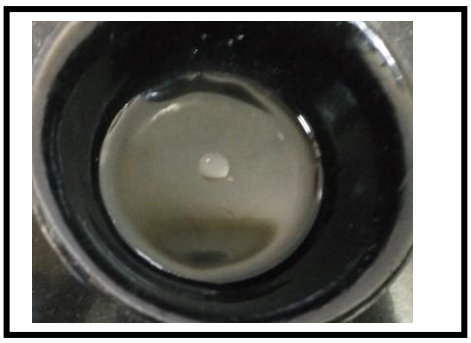
INTERPRETATION:

This patient was a known case of type 2 diabetes mellitus with Albuminuria (micro Albuminuria) , there was n urinary tract infection or any associated pitha disease. Patient showed pitham in Neikkuri on third day observation also.

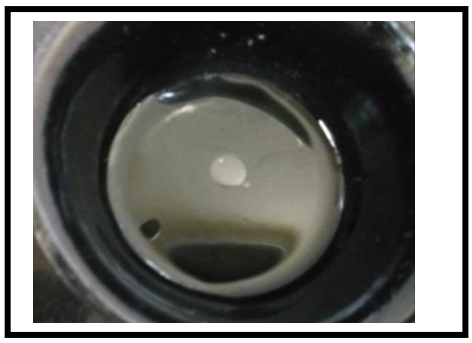
CASE 5

Neikkuri Day 1

At the moment

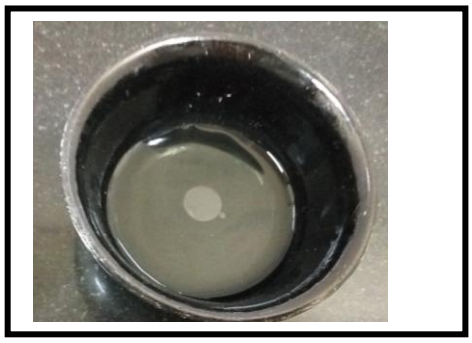


I Minute



III Minute

X Minute



Neerkkuri

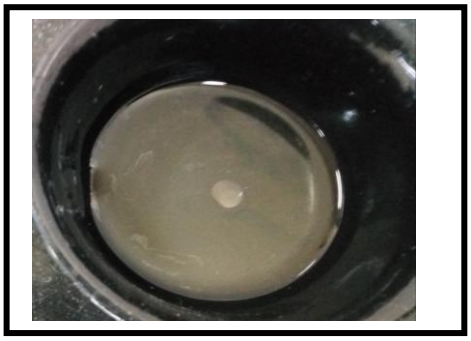
INTERPRETATION:

This patient was a known case of type 2 diabetes mellitus with Albuminuria (micro Albuminuria) , there was n urinary tract infection or any associated pitha disease. Patient showed pitham in Neikkuri on the first day observation when observed upto ten minutes.

CASE 5

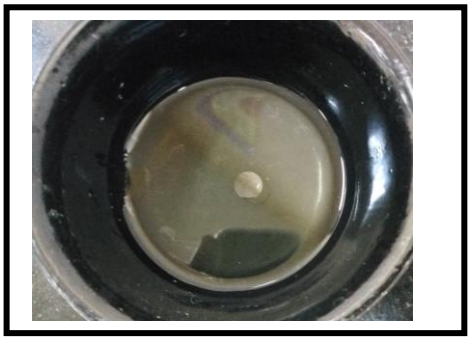
Neikkuri Day 2

At the moment

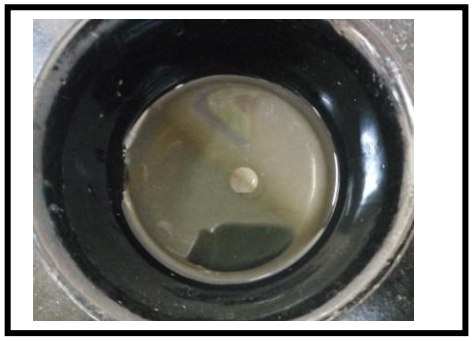


I Minute

III Minute



X Minute



Neerkkuri

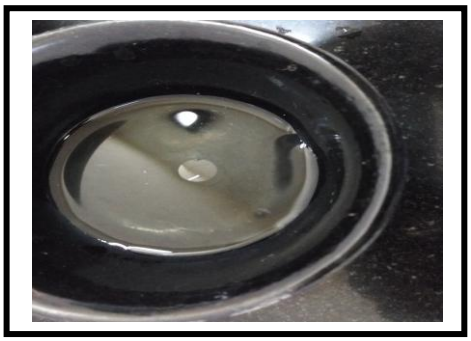
INTERPRETATION:

This patient was a known case of type 2 diabetes mellitus with Albuminuria (micro Albuminuria) , there was n urinary tract infection or any associated pitha disease. Patient showed pitham in Neikkuri on second day observation.

CASE 5

Neikkuri Day 3

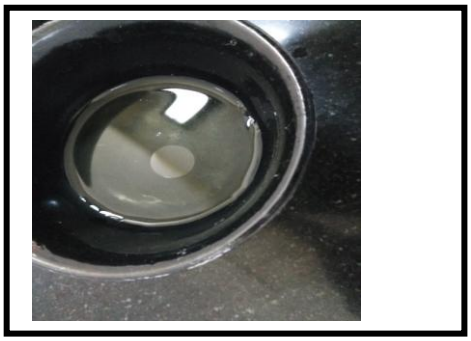
At the moment



I Minute

III Minute

X Minute



Neerkkuri

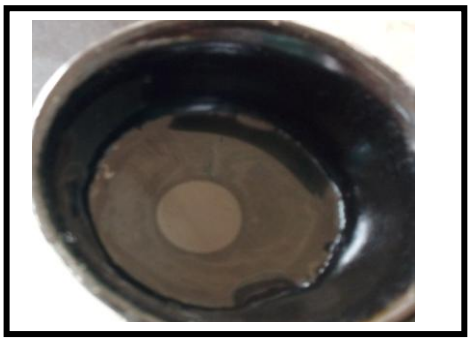
INTERPRETATION:

This patient was a known case of type 2 diabetes mellitus with Albuminuria (micro Albuminuria) , there was n urinary tract infection or any associated pitha disease. Patient showed pitham in Neikkuri on third day observation also.

CASE 6

Neikkuri Day 1

At the moment



I Minute

III Minute

X Minute

Neerkkuri

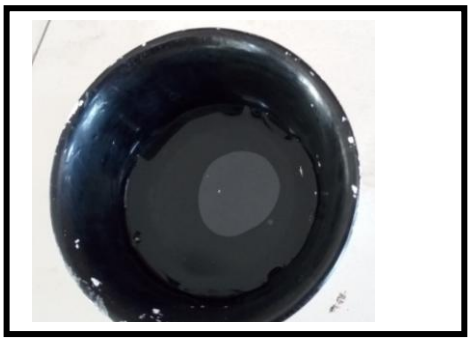
INTERPRETATION:

This patient was not a known case of type 2 diabetes mellitus , but had Urinary Tract Infection with Albuminuria (micro Albuminuria) ,since UTI also comes under megaha rogam deranged humour in pitham, that may be the reason behind pithathil pitham Neikkuri in this case.

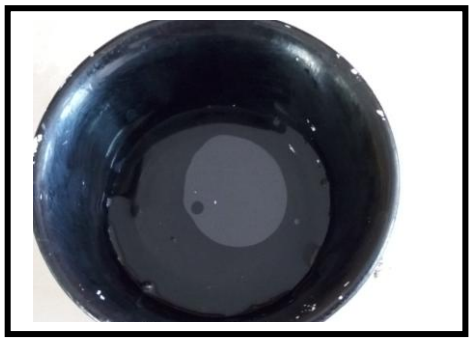
CASE 6

Neikkuri Day 2

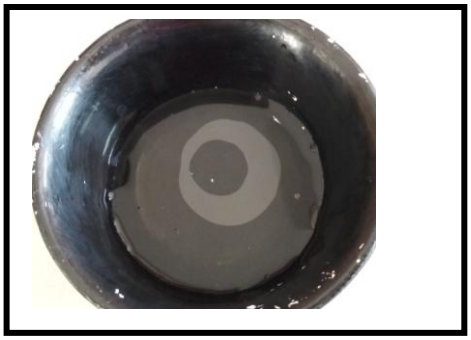
At the moment



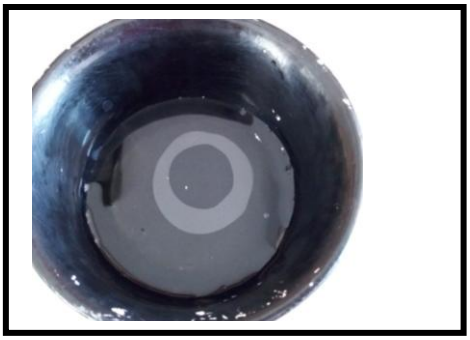
I Minute



III Minute



X Minute



Neerkkuri

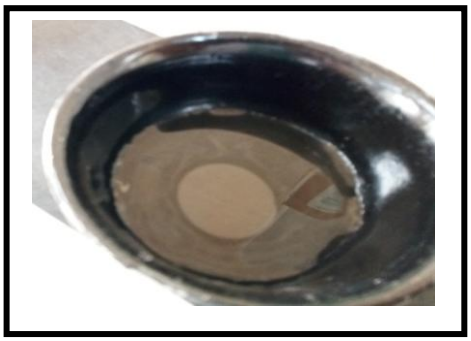
INTERPRETATION:

On the second day also patient showed pithathil pitham neikkuri.

CASE 6

Neikkuri Day 3

At the moment



I Minute

III Minute

X Minute

Neerkkuri

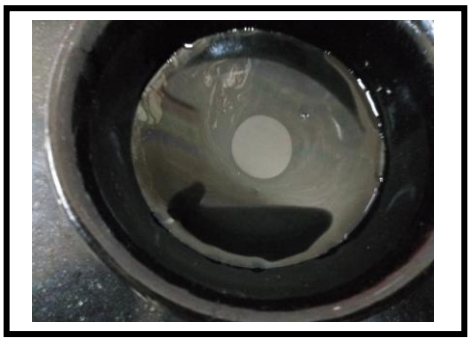
INTERPRETATION:

On the third day also patient showed piththil pitham neikkuri , which justifies th e literature “Pakar pitha vinthai alathu megham varathu”.

CASE 7

Neikkuri Day 1

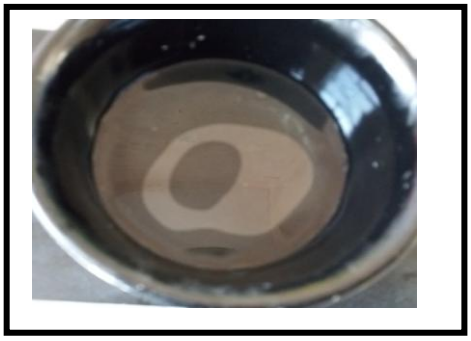
At the moment



I Minute

III Minute

X Minute



Neerkkuri

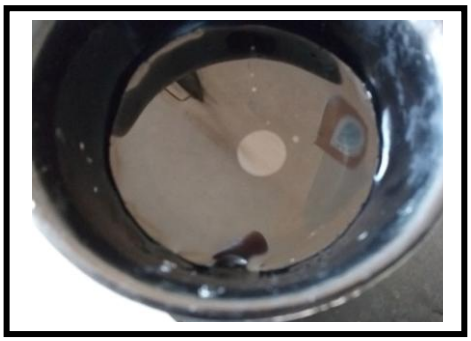
INTERPRETATION:

This patient was a known case of Type 2 diabetes with macro albuminuria and elevated serum creatinine level, all these shows elevation of pitham, and that may be the reason for pithathil pitham Neikkuri pattern.

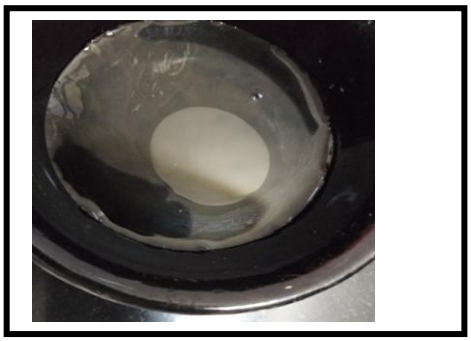
CASE 7

Neikkuri Day 2

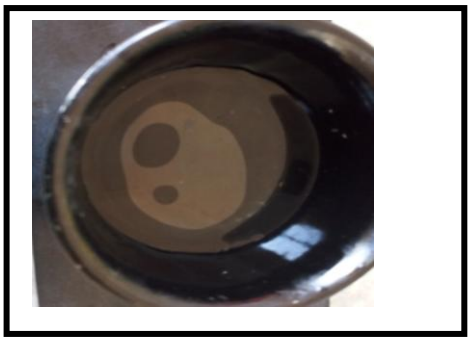
At the moment



I Minute



III Minute



X Minute

Neerkkuri

INTERPRETATION:

This patient was a known case of Type 2 diabetes with macro albuminuria and elevated serum creatinine level, all these shows elevation of pitham, and that may be the reason for pithathil pitham Neikkuri pattern and the same pattern on the second day observation helps in assuring the first day observation.

CASE 7

Neikkuri Day 3

At the moment

I Minute

III Minute

X Minute

Neerkkuri

INTERPRETATION:

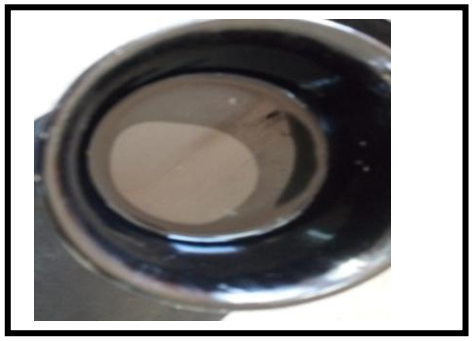
This patient was a known case of Type 2 diabetes with macro albuminuria and elevated serum creatinine level, all these shows elevation of pitham, and that may be the reason for pithathil pitham Neikkuri pattern and the same pattern on the third day observation helps in assuring the first day and second day observation.

CASE 8

Neikkuri Day 1

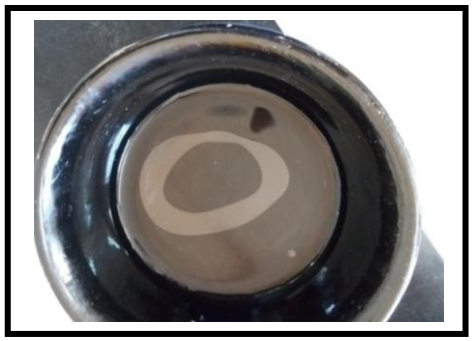
At the moment

I Minute



III Minute

X Minute



Neerkkuri

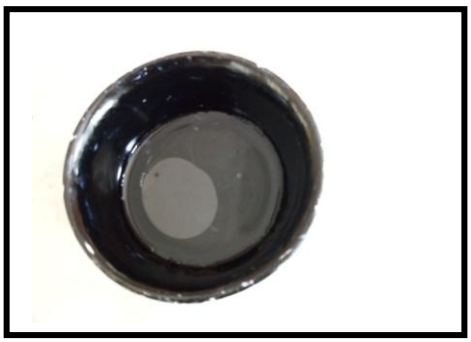
INTERPRETATION

This patient was a known case of T2 DM, urine examination showed macroalbuminuria and creatinine level was slightly elevated , that may be the reason for pithathil pitham Neikkuri pattern in this case.

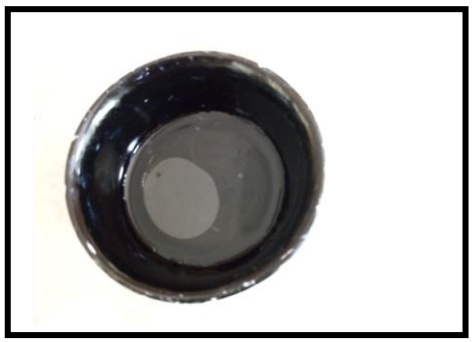
CASE 8

Neikkuri Day 2

At the moment



I Minute



III Minute

X Minute

Neerkkuri

INTERPRETATION

This patient was a known case of T2 DM, urine examination showed macroalbuminuria and creatinine level was slightly elevated , that may be the reason for pithathil pitham Neikkuri pattern in this case on second day also.

CASE 8

Neikkuri Day 3

At the moment

I Minute

III Minute

X Minute

Neerkkuri

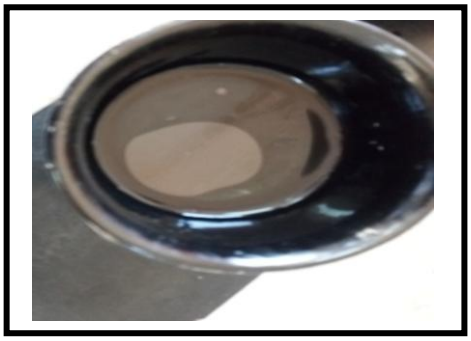
INTERPRETATION

On the third day also Neikkuri pattern was same as that of first on second day.

CASE 9

Neikkuri - Day 1

At the moment



I Minute

III Minute

X Minute

Neerkkuri

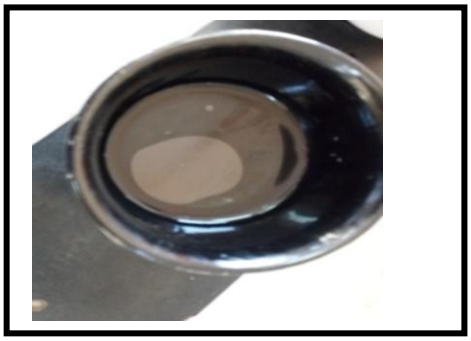
INTERPRETATION

This patient was a known case of T2 DM, urine examination showed macroalbuminuria, that may be the reason for pithathil pitham Neikkuri pattern in this case.

CASE 9

Neikkuri - Day 2

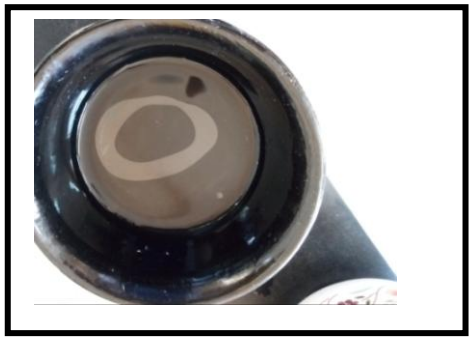
At the moment



I Minute

III Minute

X Minute



Neerkkuri

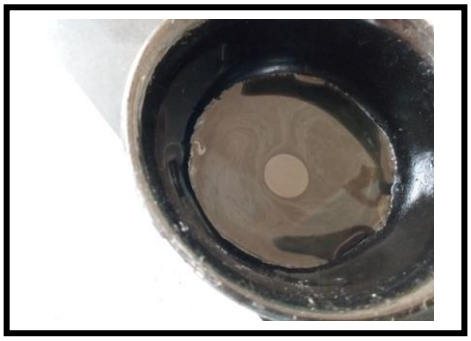
INTERPRETATION

This patient was a known case of T2 DM, urine examination showed macroalbuminuria, that may be the reason for pithathil pitham Neikkuri pattern in this case on second day also.

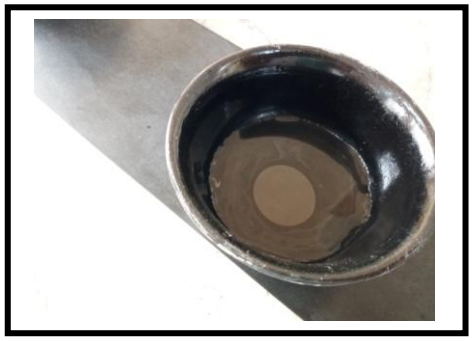
CASE 9

Neikkuri - Day 3

At the moment

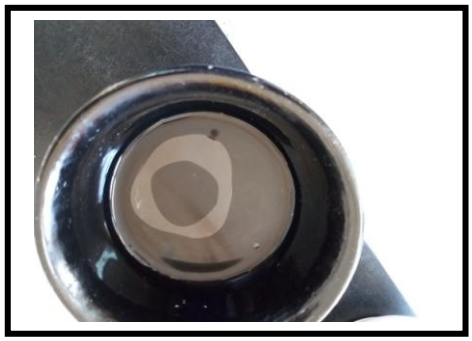


I Minute



III Minute

X Minute



Neerkkuri

INTERPRETATION

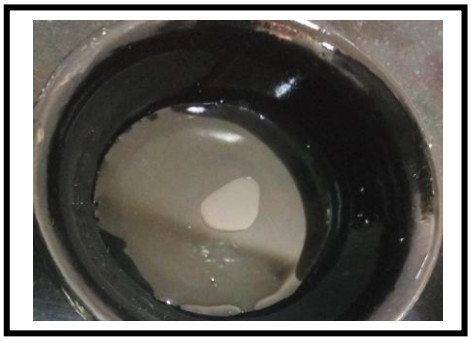
This patient was a known case of T2 DM, urine examination showed macroalbuminuria, that may be the reason for pithathil pitham Neikkuri pattern in this case on third day also.

CASE 10

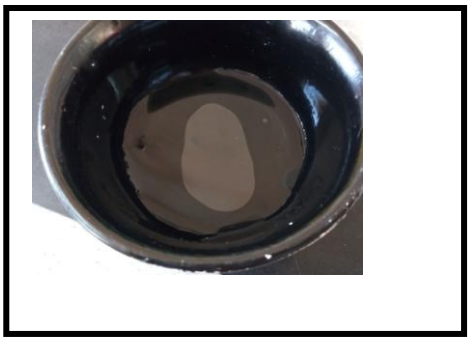
Neikkuri Day 1

At the moment

I Minute



III Minute



X Minute

Neerkkuri

INTERPRETATION

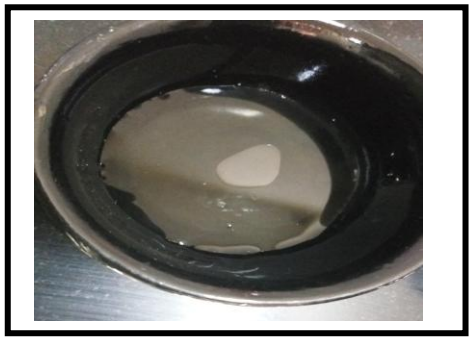
This patient was a known case of T2 DM and chronic eczema, urine examination showed microalbuminuria and plenty of bacteria. Kaba vatham Neikkuri pattern may be due to the underlying chronic disease.

CASE 10

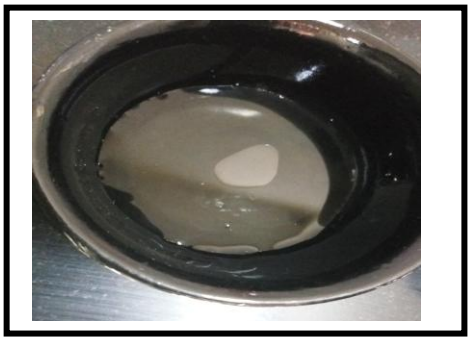
Neikkuri Day 2

At the moment

I Minute



III Minute



X Minute

Neerkkuri

INTERPRETATION

This patient was a known case of T2 DM and chronic eczema, urine examination showed microalbuminuria and plenty of bacteria. Kaba vatham Neikkuri pattern may be due to the underlying chronic disease on second day observation also.

CASE 10

Neikkuri Day 3

At the moment

I Minute

III Minute

X Minute

Neerkkuri

INTERPRETATION

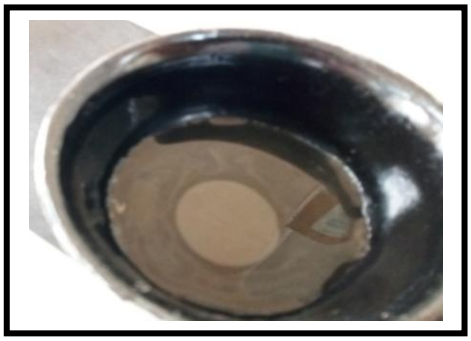
This patient was a known case of T2 DM and chronic eczema, urine examination showed microalbuminuria and plenty of bacteria. Kaba vatham Neikkuri pattern may be due to the underlying chronic disease on third day observation also.

CASE 11

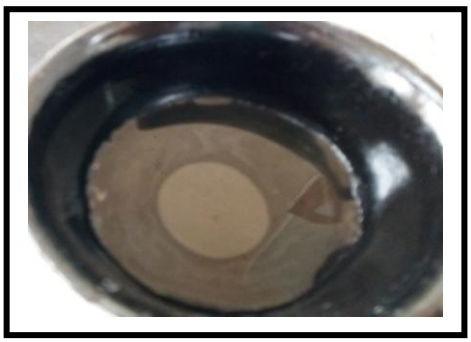
Neikkuri Day - 1

At the moment

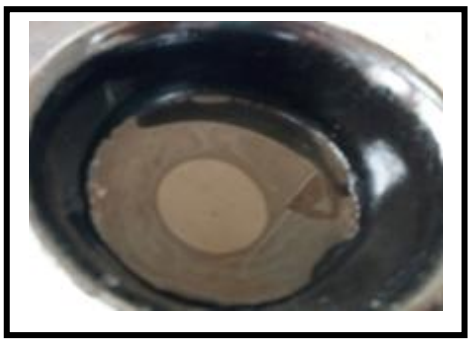
I Minute



III Minute



X Minute



Neerkkuri

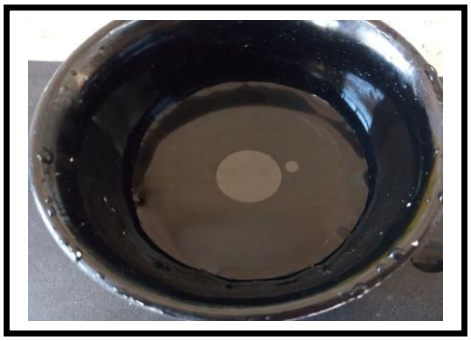
INTERPRETATION

Patient was a not known case of Diabetes Mellitus but had burning micturition and on urine examination it showed micro albuminuria, that may be the reason of pitham pattern when neikkuri was observed.

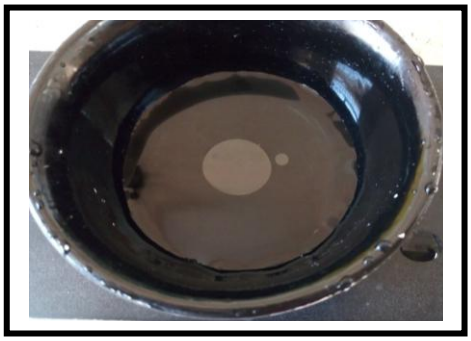
CASE 11

Neikkuri - Day -2

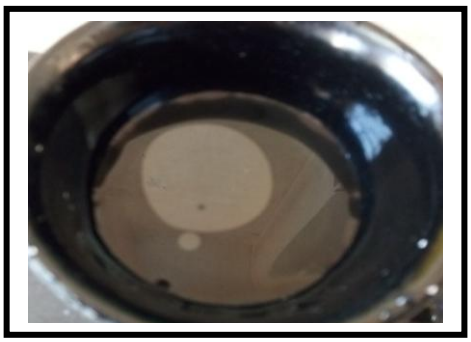
At the moment



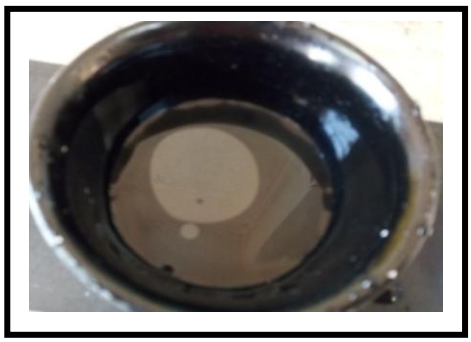
I Minute



III Minute



X Minute



Neerkkuri

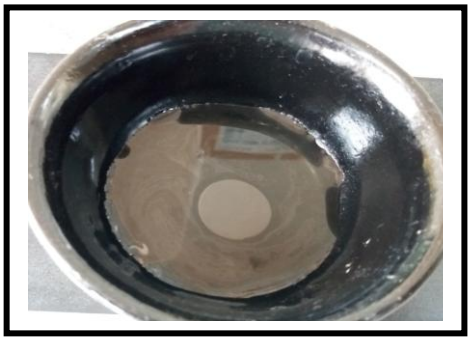
INTERPRETATION

Patient was a not known case of Diabetes Mellitus but had burning micturition and on urine examination it showed micro albuminuria, which may be the reason of pitham pattern when neikkuri was observed even on second day.

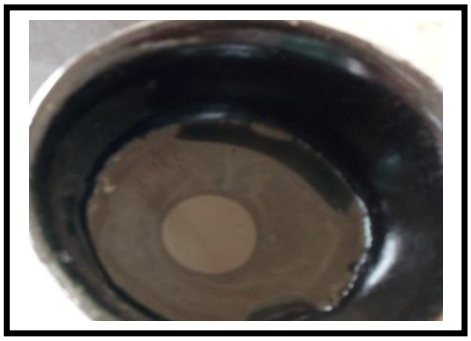
CASE II

Neikkuri Day 3

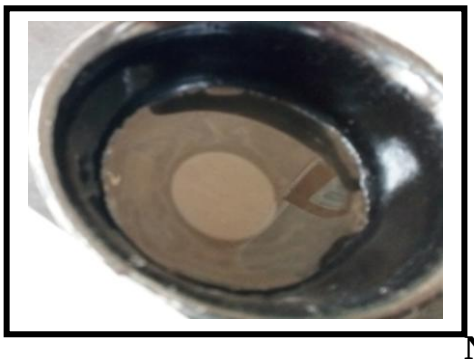
At the moment



I Minute



III Minute



X Minute

Neerkkuri

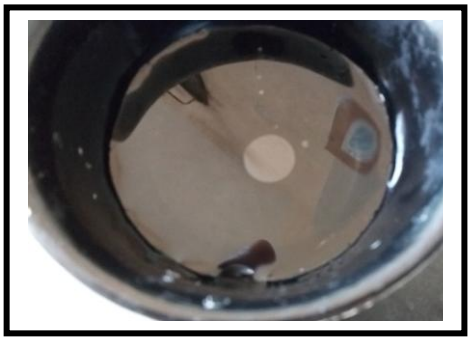
INTERPRETATION

Patient was a not known case of Diabetes Mellitus but had burning micturition and on urine examination it showed micro albuminuria, which may be the reason of pitham pattern when neikkuri was observed even on third day also.

CASE 12

Neikkuri Day 1

At the moment



I Minute

III Minute

X Minute

Neerkkuri

I**NTERPRETATION**

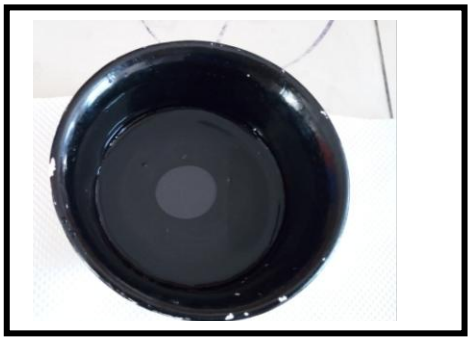
Patient was a known case of Diabetes Mellitus on urine examination it showed micro albuminuria, which may be the reason of pitham pattern when neikkuri was observed even on first day.

CASE 12

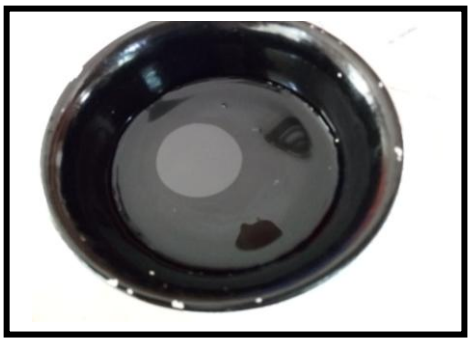
Neikkuri – Day - 2

At the moment

I Minute



III Minute



X Minute

Neerkkuri

INTERPRETATION

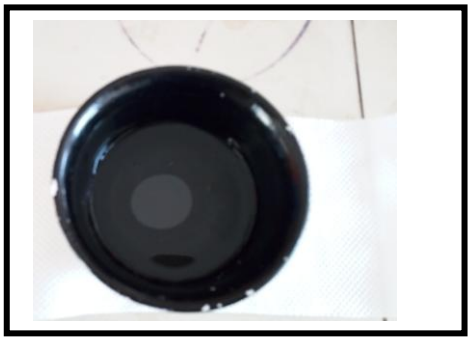
Patient was a known case of Diabetes Mellitus on urine examination it showed micro albuminuria, which may be the reason of pitham pattern when neikkuri was observed even on second day.

CASE 12

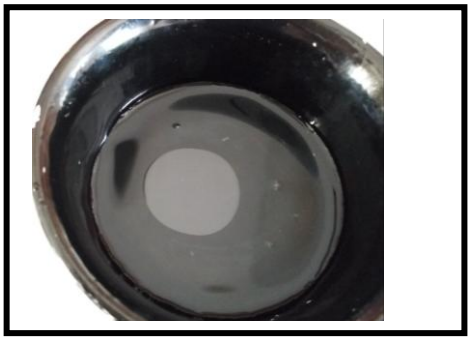
Neikkuri Day - 3

At the moment

I Minute



III Minute



X Minute

Neerkkuri

INTERPRETATION

Patient was a known case of Diabetes Mellitus on urine examination it showed micro albuminuria, which may be the reason of pitham pattern when neikkuri was observed even on third day.

CASE 13

Neikkuri Day - 1

At the moment

I Minute

III Minute

X Minute

Neerkkuri

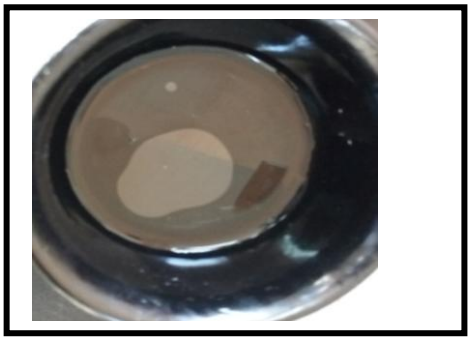
INTERPRETATION

Patient was a known case of Diabetes Mellitus on urine examination it showed macro albuminuria, which may be the reason of pithathil pitham pattern when neikkuri was observed even on first day.

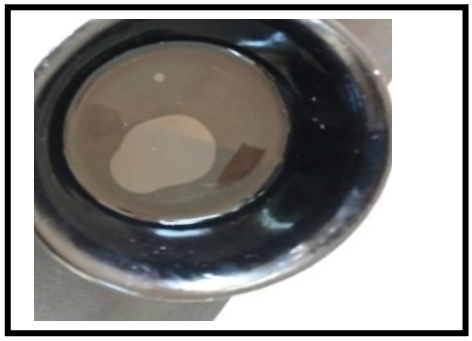
CASE 13

Neikkuri Day- 2

At the moment



I Minute



III Minute

X Minute

Neerkkuri

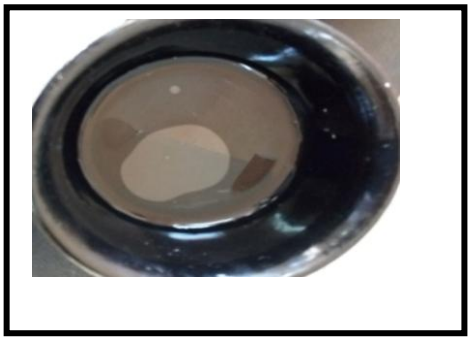
INTERPRETATION

Patient was a known case of Diabetes Mellitus on urine examination it showed macro albuminuria, which may be the reason of pithathil pitham pattern when neikkuri was observed even on second day.

CASE 13

Neikkuri - Day -3

At the moment



I Minute

III Minute

X Minute

Neerkkuri

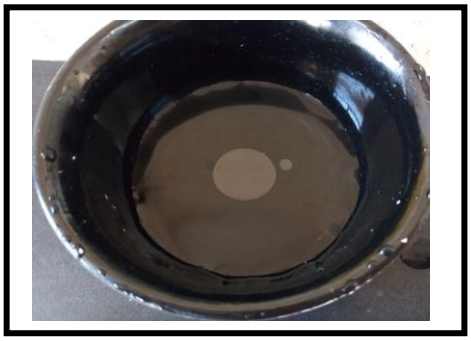
INTERPRETATION

Patient was a known case of Diabetes Mellitus on urine examination it showed macro albuminuria, which may be the reason of pithathil pitham pattern when neikkuri was observed even on third day.

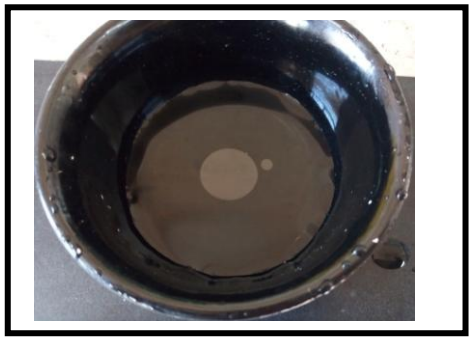
CASE 14

Neikkuri Day- 1

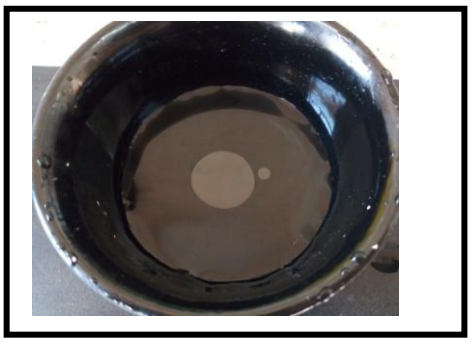
At the moment



I Minute



III Minute



X Minute

Neerkkuri

INTERPRETATION

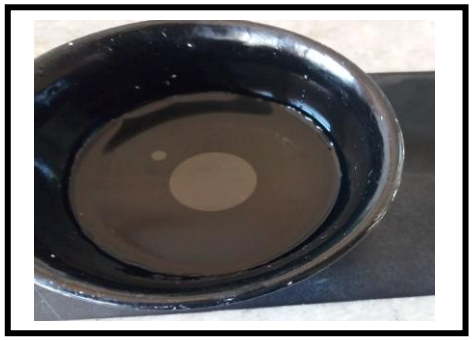
Patient was a known case of Diabetes Mellitus on urine examination it showed micro albuminuria, which may be the reason of pitham pattern when neikkuri was observed .

CASE 14

Neikkuri Day 2

At the moment

I Minute



III Minute

X Minute

Neerkkuri

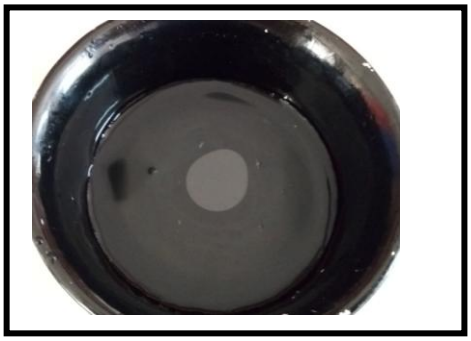
INTERPRETATION

Patient was a known case of Diabetes Mellitus on urine examination it showed micro albuminuria, which may be the reason of pitham pattern when neikkuri was observed on second day also.

CASE 14

Neikkuri Day- 3

At the moment



I Minute

III Minute

X Minute

Neerkkuri

INTERPRETATION

Patient was a known case of Diabetes Mellitus on urine examination it showed micro albuminuria, which may be the reason of pitham pattern when neikkuri was observed on third day also.

CASE 15

Neikkuri –Day- 1

At the moment

I Minute

III Minute

X Minute

Neerkkuri

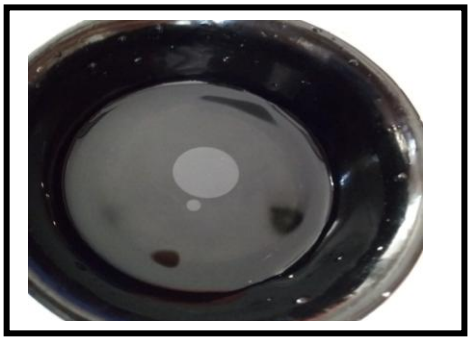
INTERPRETATION

Patient was a known case of Diabetes Mellitus on urine examination it showed micro albuminuria, which may be the reason of pitham pattern when neikkuri was observed.

CASE 15

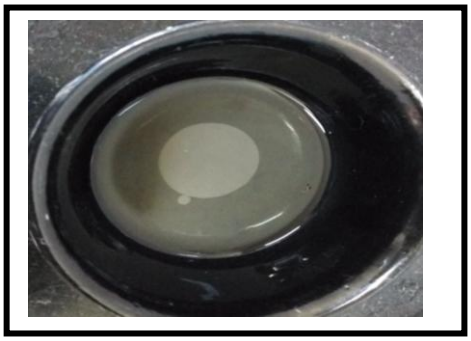
Neikkuri -Day 2

At the moment

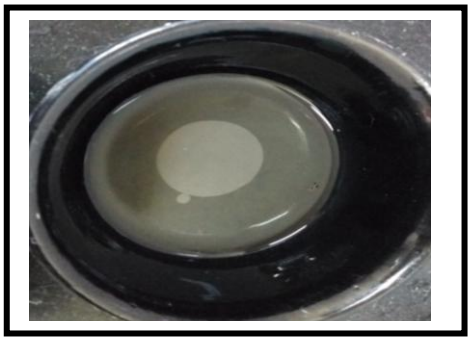


I Minute

III Minute



X Minute



Neerkkuri

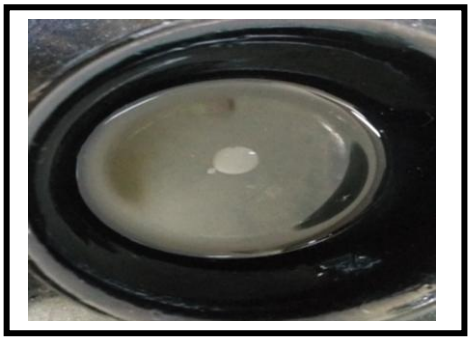
INTERPRETATION

Patient was a known case of Diabetes Mellitus on urine examination it showed micro albuminuria, which may be the reason of pitham pattern when neikkuri was observed on day two.

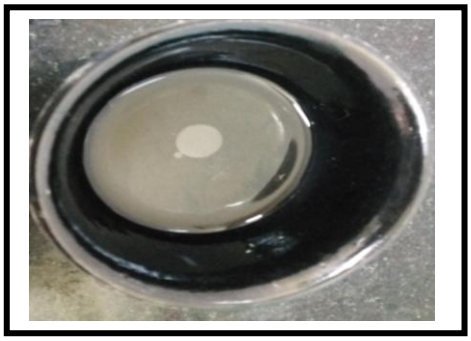
CASE 15

Neikkuri -Day 3

At the moment

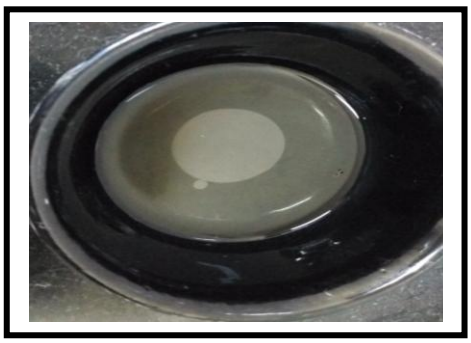


I Minute



III Minute

X Minute



Neerkkuri

INTERPRETATION

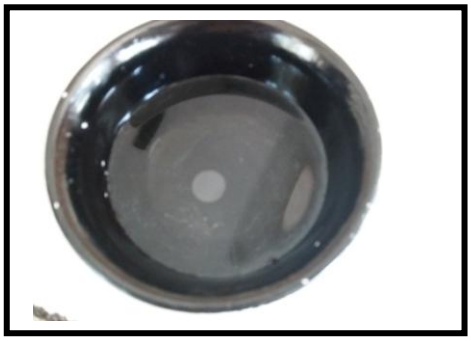
Patient was a known case of Diabetes Mellitus on urine examination it showed micro albuminuria, which may be the reason of pitham pattern when neikkuri was observed on day three.

CASE 16

Neikkuri -Day 1

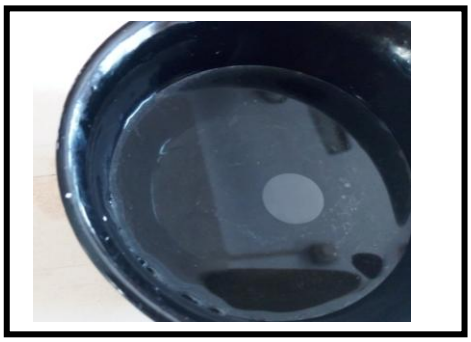
At the moment

I Minute



III Minute

X Minute



Neerkkuri

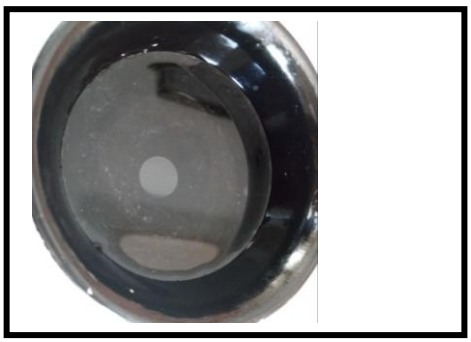
INTERPRETATION

This patient was a known case of Diabetes mellitus, urine examination showed micro albuminuria that may be the reason for pitham pattern in Neikkuri observation.

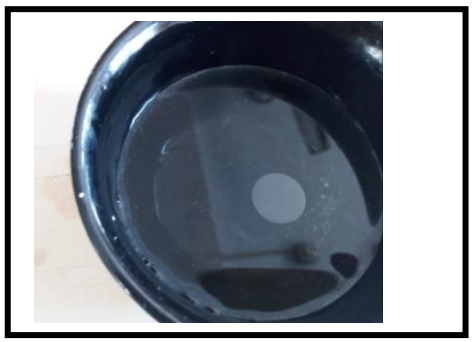
CASE 16

Neikkuri Day- 2

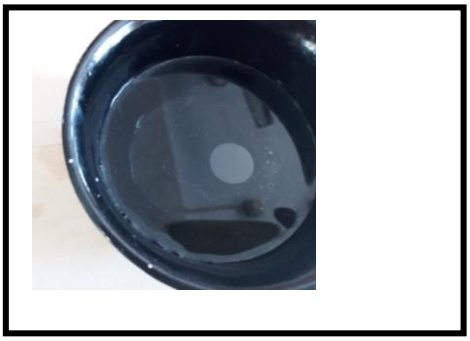
At the moment



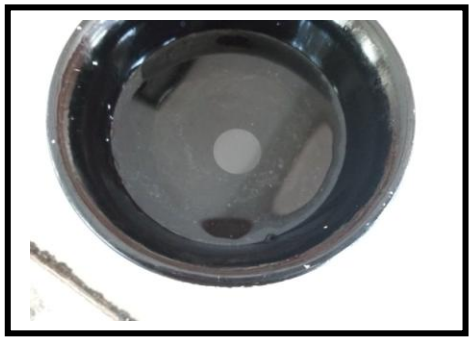
I Minute



III Minute



X Minute



Neerkkuri

INTERPRETATION

This patient was a known case of Diabetes mellitus, urine examination showed micro albuminuria that may be the reason for pitham pattern in Neikkuri observation even on second day.

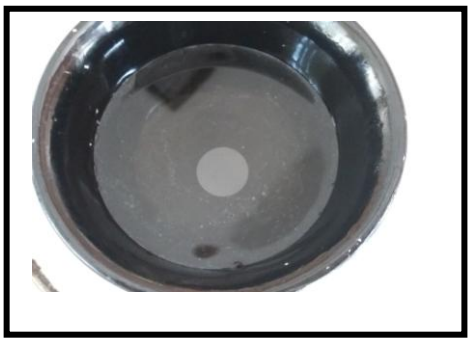
CASE 16

Neikkuri - Day 3

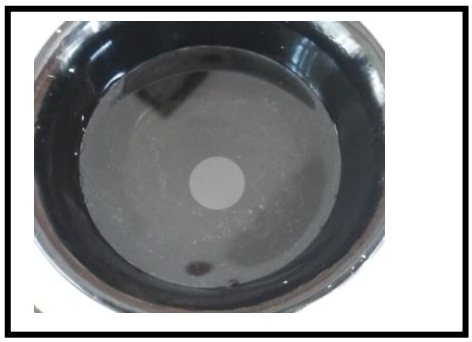
At the moment

I Minute

III Minute



X Minute



Neerkkuri

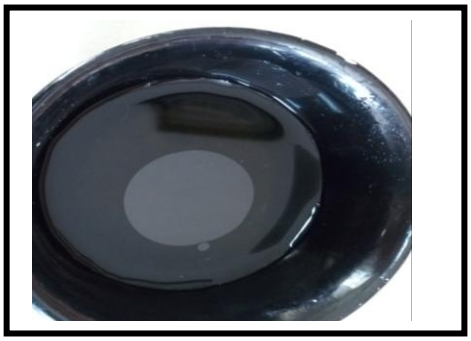
INTERPRETATION

This patient was a known case of Diabetes mellitus, urine examination showed micro albuminuria that may be the reason for pitham pattern in Neikkuri observation even on third day.

CASE 17

Neikkuri -Day 1

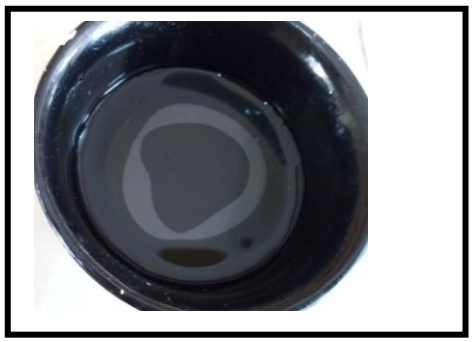
At the moment



I Minute

III Minute

X Minute



Neerkkuri

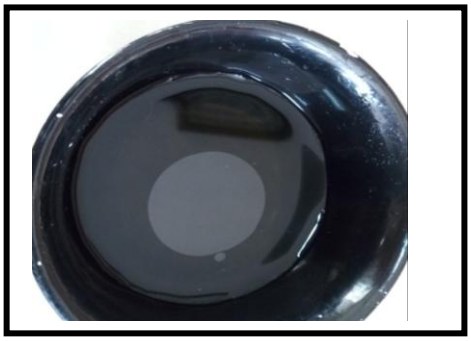
INTERPRETATION

This patient was a known case of Diabetes Mellitus and on urine examination showed micro Albuminuria, on Neikkuri observation pithathil pitham was shown which indicates the derangement of pitham .

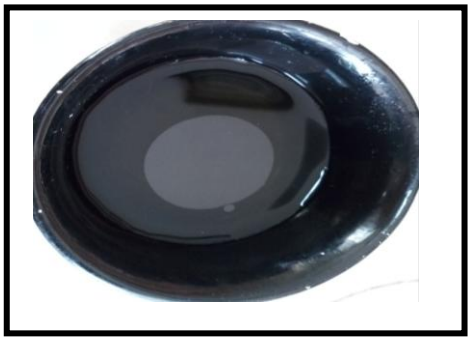
CASE 17

Neikkuri -Day 2

At the moment

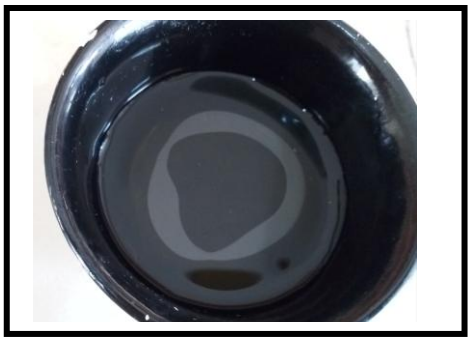


I Minute



III Minute

X Minute



Neerkkuri

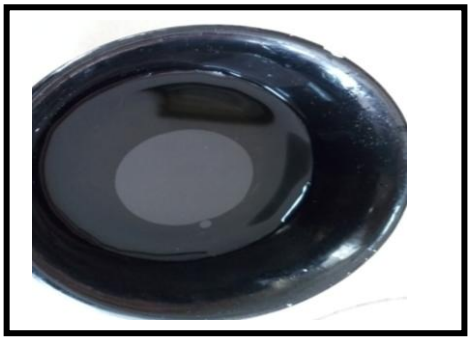
INTERPRETATION

This patient was a known case of Diabetes Mellitus and on urine examination showed micro Albuminuria, on Neikkuri observation pithathil pitham was shown which indicates the derangement of pitham even on second day .

CASE 17

Neikkuri Day- 3

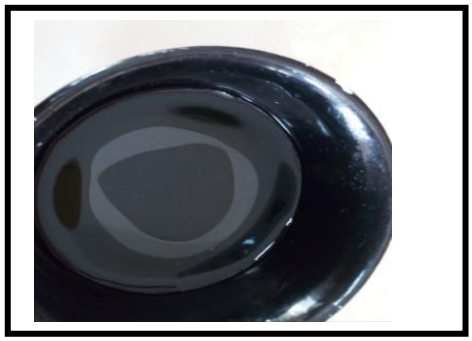
At the moment



I Minute

III Minute

X Minute



Neerkkuri

INTERPRETATION

This patient was a known case of Diabetes Mellitus and on urine examination showed micro Albuminuria, on Neikkuri observation pithathil pitham was shown which indicates the derangement of pitham even on third day .

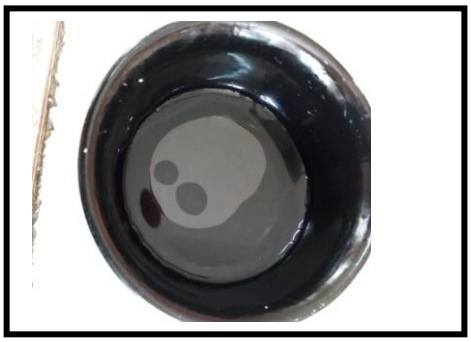
CASE 18

Neikkuri - Day 1

At the moment

I Minute

III Minute



X Minute

Neerkkuri

INTERPRETATION

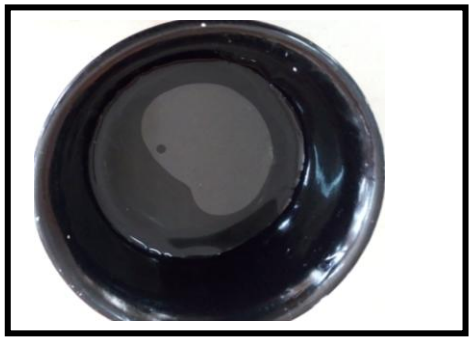
This patient was a known case of Diabetes Mellitus creatinine was elevated when blood investigation was done, on Neikkuri observation pithathil pitham was shown which indicates the derangement of pitham.

CASE 18

Neikkuri -Day 2

At the moment

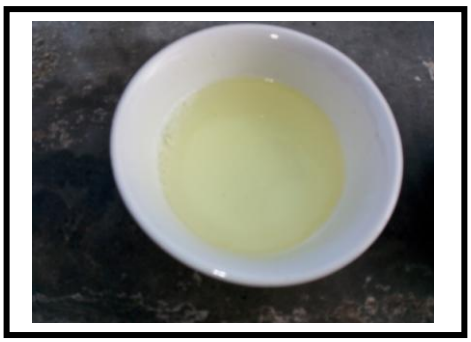
I Minute



III Minute

X Minute

Neerkkuri



INTERPRETATION

This patient was a known case of Diabetes Mellitus creatinine was elevated when blood investigation was done, on Neikkuri observation pithathil pitham was shown even on second day which indicates the derangement of pitham .

CASE 18

Neikkuri -Day 3

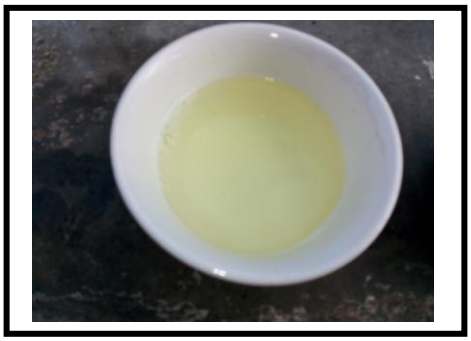
At the moment

I Minute

III Minute

X Minute

Neerkkuri



INTERPRETATION

This patient was a known case of Diabetes Mellitus creatinine was elevated when blood investigation was done, on Neikkuri observation pithathil pitham was shown even on third day which indicates the derangement of pitham.

CASE 19

Neikkuri -Day 1

At the moment

I Minute

III Minute

X Minute

Neerkkuri

INTERPRETATION

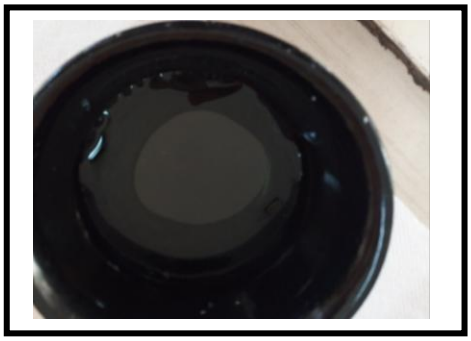
This patient was a known case of Diabetes Mellitus, on Neikkuri observation pitham pattern was shown even on first day which indicates the derangement of pitham .

CASE 19

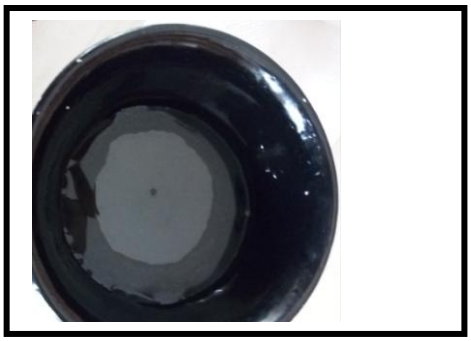
Neikkuri -Day 2

At the moment

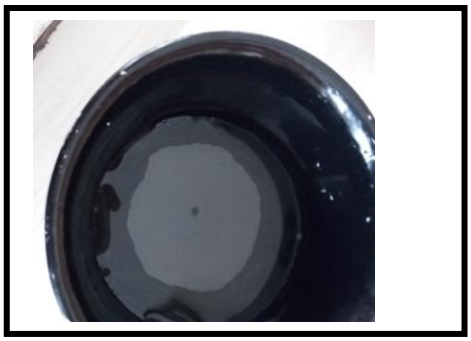
I Minute



III Minute



X Minute



Neerkkuri

INTERPRETATION

This patient was a known case of Diabetes Mellitus, on Neikkuri observation pitham pattern was shown even on second day which indicates the derangement of pitham .

CASE 19

Neikkuri -Day 3

At the moment

I Minute

III Minute

X Minute

Neerkkuri

INTERPRETATION

This patient was a known case of Diabetes Mellitus, on Neikkuri observation pitham pattern was shown even on third day which indicates the derangement of pitham .

CASE 20

Neikkuri Day 1

At the moment

I Minute

III Minute

X Minute

Neerkkuri

INTERPRETATION

This patient was a known case of Diabetes Mellitus,on urine examination macro Albuminuria was present as well as bacteria was loaded ; on Neikkuri observation pitham pattern was shown on first day which indicates the derangement of pitham

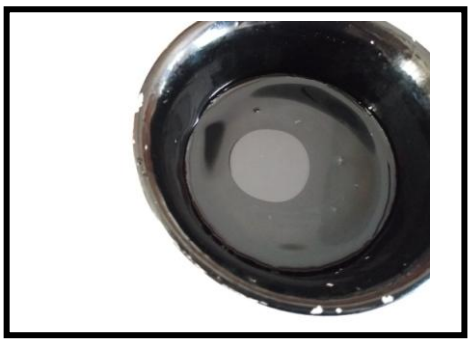
CASE 20

Neikkuri -Day 2

At the moment

I Minute

III Minute



X Minute

Neerkkuri

INTERPRETATION

This patient was a known case of Diabetes Mellitus,on urine examination macro Albuminuria was present as well as bacteria was loaded; on Neikkuri observation pitham pattern was shown on second day which indicates the derangement of pitham .

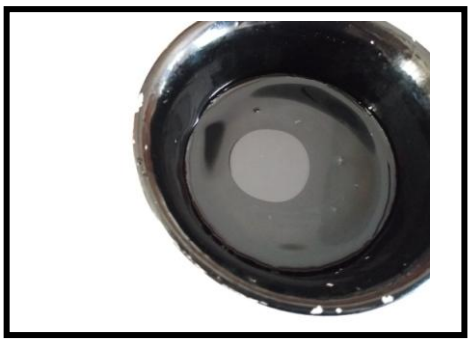
CASE 20

Neikkuri -Day 3

At the moment

I Minute

III Minute



X Minute

Neerkkuri

INTERPRETATION

This patient was a known case of Diabetes Mellitus,on urine examination macro Albuminuria was present as well as bacteria was loaded ; on Neikkuri observation pitham pattern was shown on third day which indicates the derangement of pitham .

CASE 21

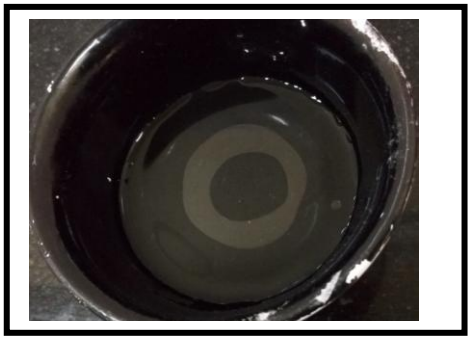
Neikkuri -Day 1

At the moment

I Minute

III Minute

X Minute



Neerkkuri

INTERPRETATION

This patient was a known case of Diabetes Mellitus, Benign Prostatic Hypertrophy on urine examination micro albuminuria was present ; on Neikkuri observation pithathil pitham pattern was shown on first day since both these conditions are due to derangement of pitha this urine examination justifies the tridosha principles.

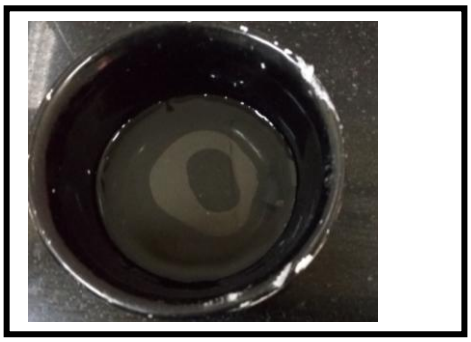
CASE 21

Neikkuri -Day 2

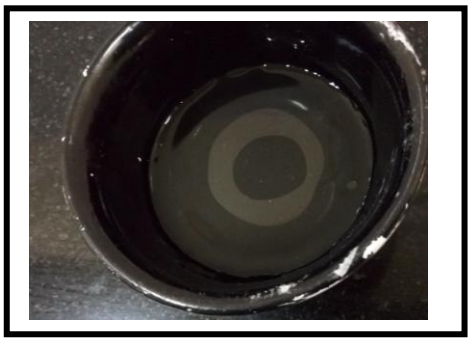
At the moment

I Minute

III Minute



X Minute



Neerkkuri

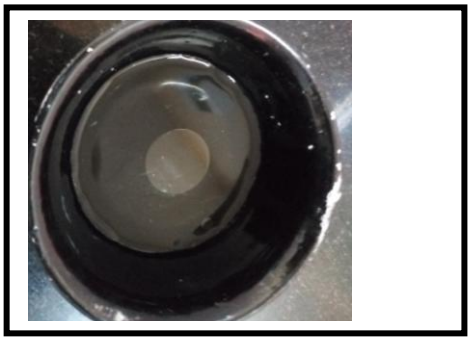
INTERPRETATION

This patient was a known case of Diabetes Mellitus, Benign Prostatic Hypertrophy on urine examination micro albuminuria was present ; on Neikkuri observation pithathil pitham pattern was shown even on second day since both these conditions are due to derangement of pitha this urine examination justifies the tridosha principles.

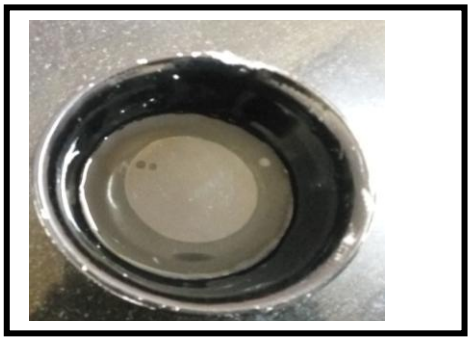
CASE 21

Neikkuri Day- 3

At the moment

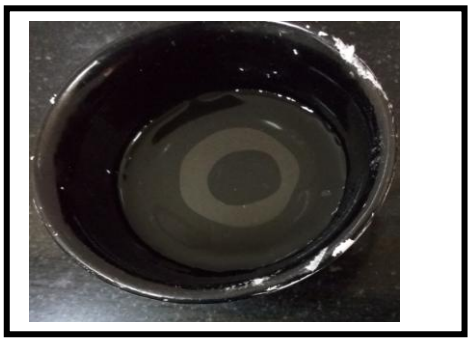


I Minute



III Minute

X Minute



Neerkkuri

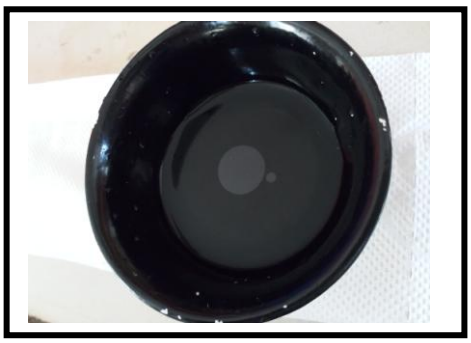
INTERPRETATION

This patient was a known case of Diabetes Mellitus, Benign Prostatic Hypertrophy on urine examination micro albuminuria was present ; on Neikkuri observation pithathil pitham pattern was shown even on third day since both these conditions are due to derangement of pitha this urine examination justifies the tridosha principles.

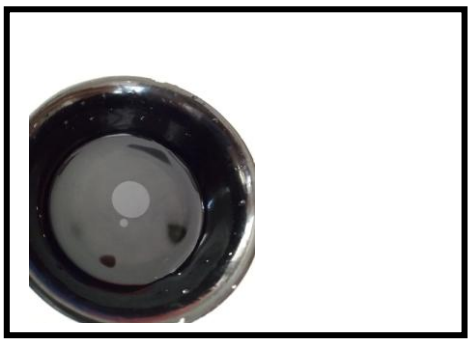
CASE 22

Neikkuri Day -1

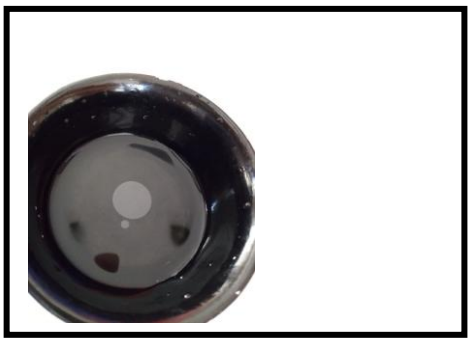
At the moment



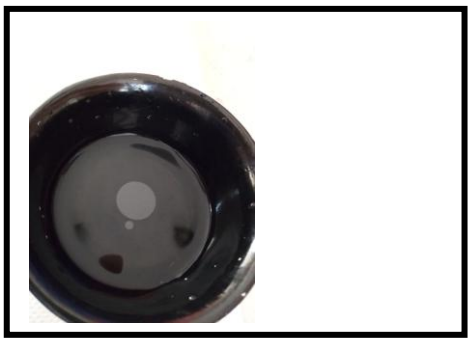
I Minute



III Minute



X Minute



Neerkkuri

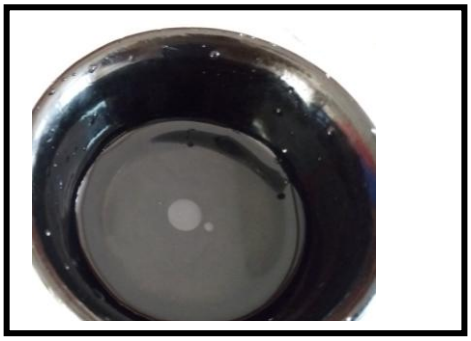
INTERPRETATION

This patient was a known case of Diabetes Mellitus,on urine examination micro albuminuria was present ; on Neikkuri observation pitham pattern was shown on first day.

CASE 22

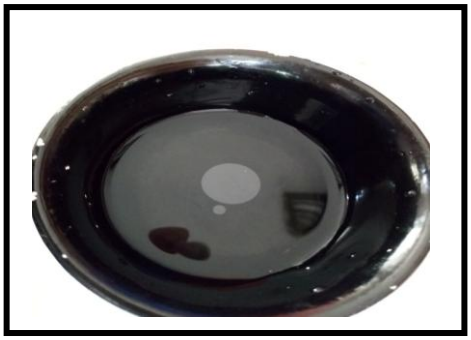
Neikkuri Day 2

At the moment



I Minute

III Minute



X Minute

Neerkkuri

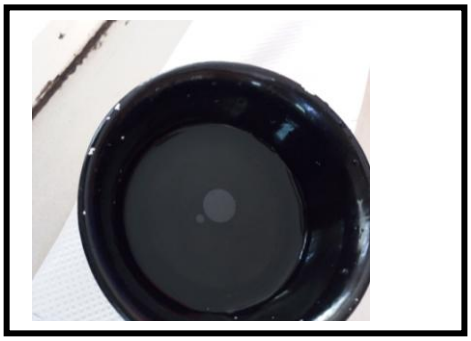
INTERPRETATION

This patient was a known case of Diabetes Mellitus,on urine examination micro albuminuria was present ; on Neikkuri observation pitham pattern was shown on second day observation.

CASE 22

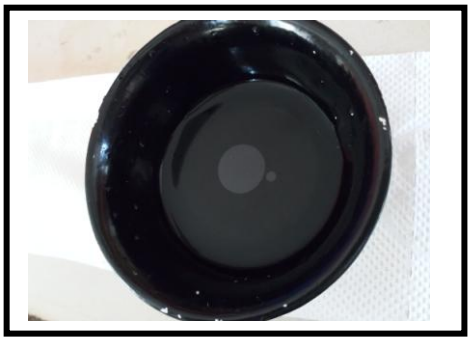
Neikkuri Day- 3

At the moment



I Minute

III Minute



X Minute

Neerkkuri

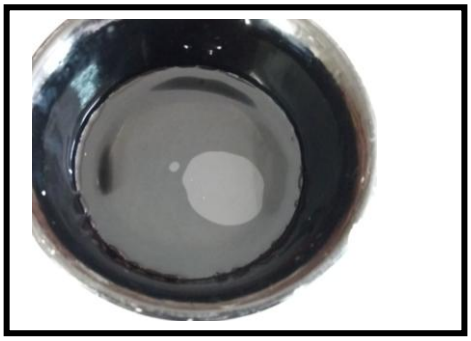
INTERPRETATION

This patient was a known case of Diabetes Mellitus,on urine examination micro albuminuria was present ; on Neikkuri observation pitham pattern was shown on third day.

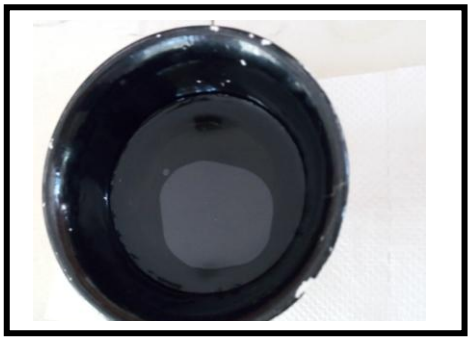
CASE 23

Neikkuri Day 1

At the moment



I Minute



III Minute

X Minute

Neerkkuri

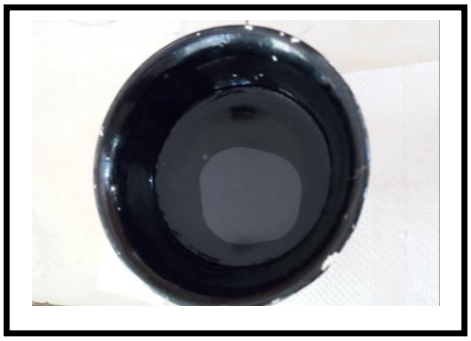
INTERPRETATION

This patient was a known case of Diabetes Mellitus,on urine examination micro albuminuria was present ; on Neikkuri observation pitham pattern was shown on first day.

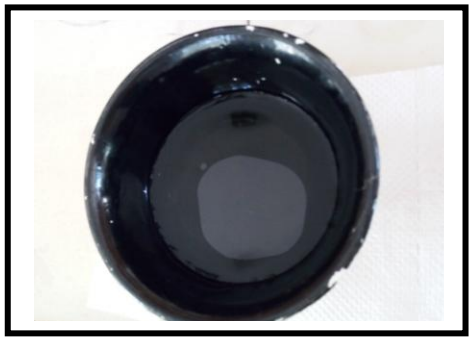
CASE 23

Neikkuri Day- 2

At the moment



I Minute



III Minute

X Minute

Neerkkuri

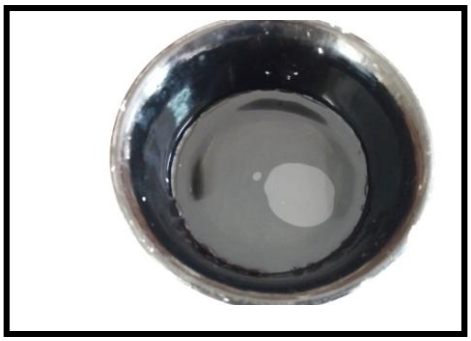
INTERPRETATION

This patient was a known case of Diabetes Mellitus,on urine examination micro albuminuria was present ; on Neikkuri observation pitham pattern was shown on second day.

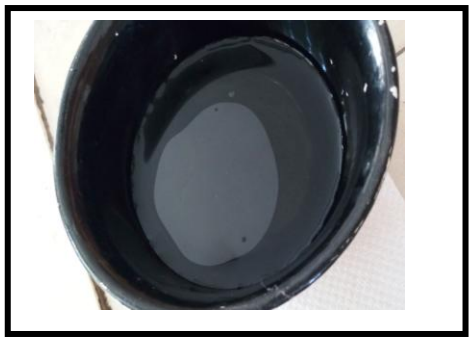
CASE 23

Neikkuri Day -3

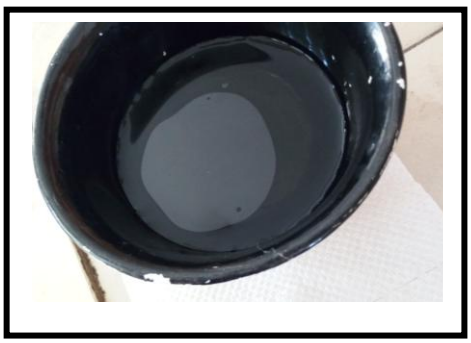
At the moment



I Minute



III Minute



X Minute

Neerkkuri

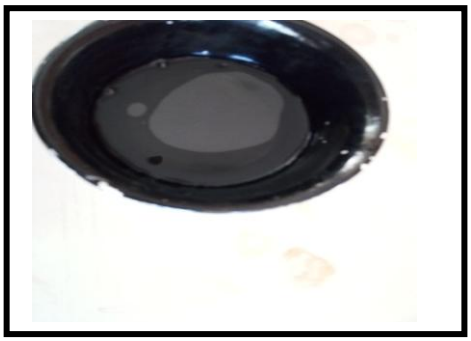
INTERPRETATION

This patient was a known case of Diabetes Mellitus ,on urine examination micro albuminuria was present ; on Neikkuri observation pitham pattern was shown on third day.

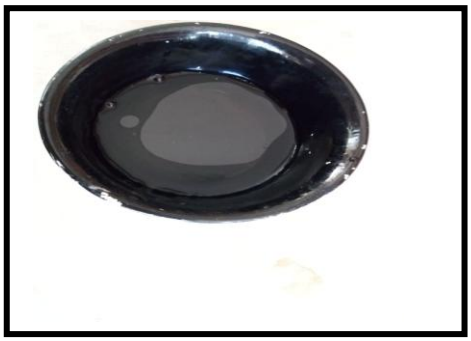
CASE 24

Neikkuri Day- 1

At the moment



I Minute



III Minute

X Minute

Neerkkuri

INTERPRETATION

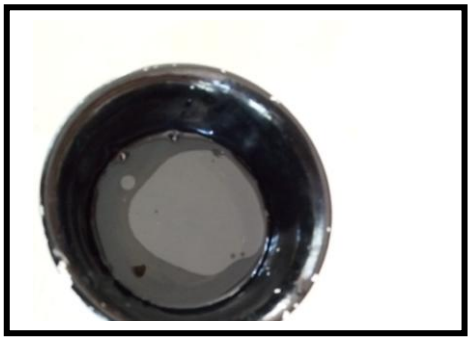
This patient was a known case of Diabetes Mellitus, on urine examination macro albuminuria was present; on Neikkuri observation pithathil pitham pattern was shown on first day.

CASE 24

Neikkuri Day- 2

At the moment

I Minute



III Minute

X Minute

Neerkkuri

INTERPRETATION

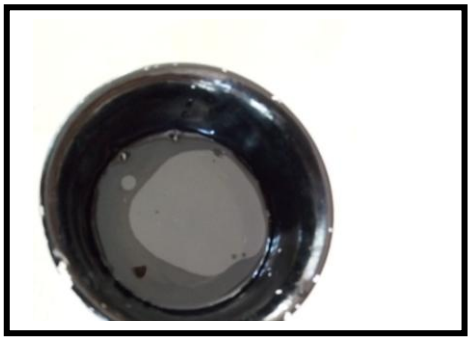
This patient was a known case of Diabetes Mellitus, on urine examination macro albuminuria was present; on Neikkuri observation pithathil pitham pattern was shown on second day.

CASE 24

Neikkuri Day- 3

At the moment

I Minute



III Minute

X Minute

Neerkkuri

INTERPRETATION

This patient was a known case of Diabetes Mellitus, on urine examination macro albuminuria was present; on Neikkuri observation pithathil pitham pattern was shown on third day.

CASE 25

Neikkuri Day- 1

At the moment

I Minute

III Minute

X Minute

Neerkkuri

INTERPRETATION

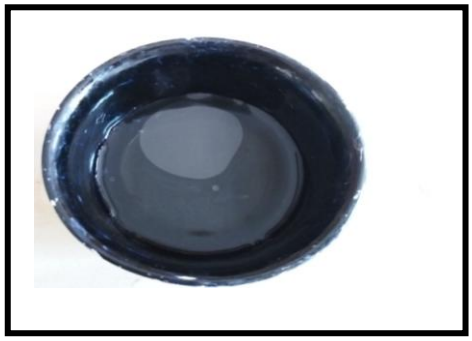
This patient was a known case of Diabetes Mellitus, on urine examination macro albuminuria was present; on Neikkuri observation pithathil pitham pattern was shown on first day.

CASE 25

Neikkuri Day- 2

At the moment

I Minute



III Minute

X Minute

Neerkkuri

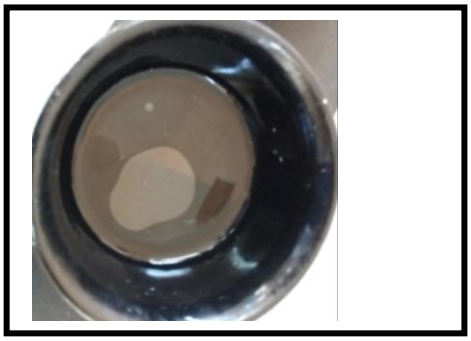
INTERPRETATION

Patient was a known case of Diabetes Mellitus and on urine examination macro Albuminuria was shown, Neikkuri examination showed pithathilpitham pattern even on second day.

CASE 25

Neikkuri Day- 3

At the moment



I Minute

III Minute

X Minute

Neerkkuri

INTERPRETATION

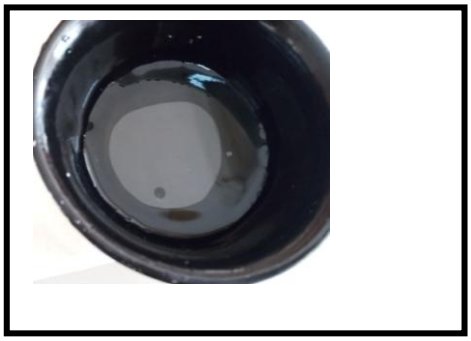
Patient was a known case of Diabetes Mellitus and on urine examination macro Albuminuria was shown, Neikkuri examination showed pithathilpitham pattern even on third day.

CASE 26

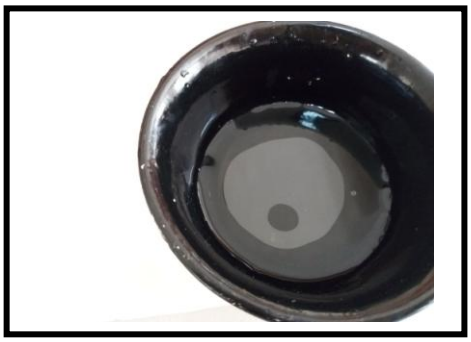
Neikkuri Day- 1

At the moment

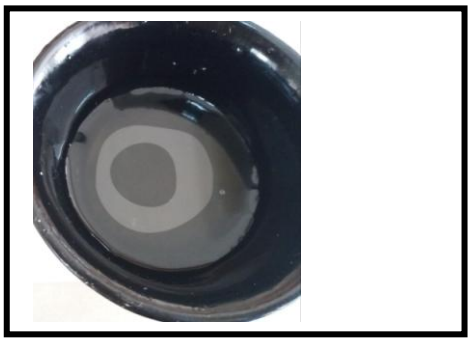
I Minute



III Minute



X Minute



Neerkkuri

INTERPRETATION

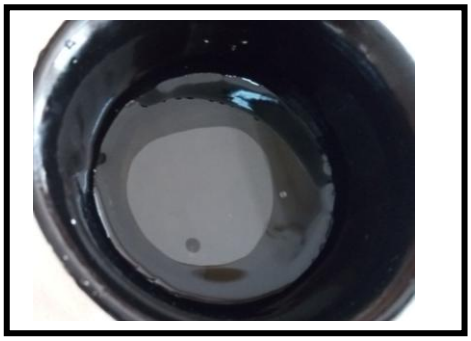
Patient was a known case of Diabetes Mellitus and on urine examination macro Albuminuria as wll as plenty of pus cells and epithelial cells were shown, Neikkuri examination showed pithathilpitham pattern on first day.

CASE 26

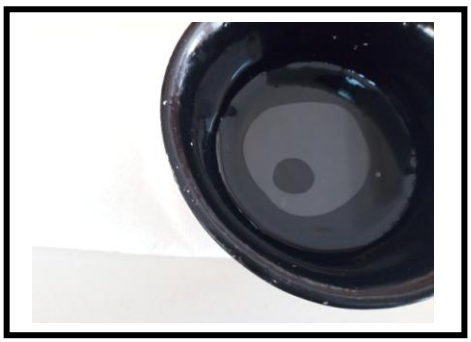
Neikkuri Day- 2

At the moment

I Minute



III Minute



X Minute

Neerkkuri

INTERPRETATION

Patient was a known case of Diabetes Mellitus and on urine examination macro Albuminuria as wll as plenty of pus cells and epithelial cells were shown, Neikkuri examination showed pithathilpitham pattern on second day.

CASE 26

Neikkuri Day -3

At the moment

I Minute

III Minute

X Minute

Neerkkuri

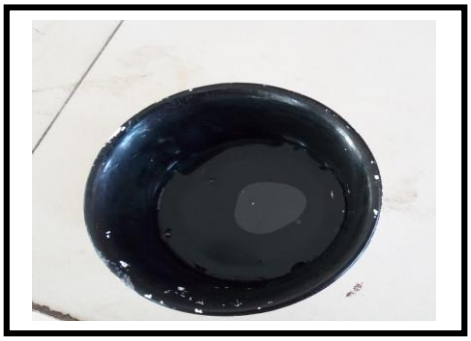
INTERPRETATION

Patient was a known case of Diabetes Mellitus and on urine examination macro Albuminuria as well as plenty of pus cells and epithelial cells were shown, Neikkuri examination showed pithathilpitham pattern on third day.

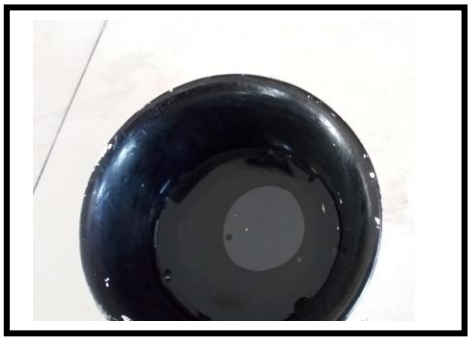
CASE 27

Neikkuri Day- 1

At the moment



I Minute



III Minute

X Minute

Neerkkuri

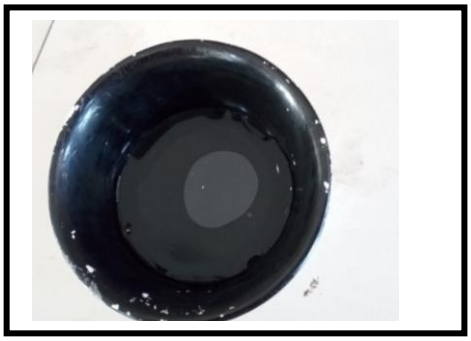
INTERPRETATION

Patient was a known case of Diabetes Mellitus and on urine examination micro Albuminuria and on blood investigation elevated creatinine were shown, Neikkuri examination showed pithathilpitham pattern on first day.

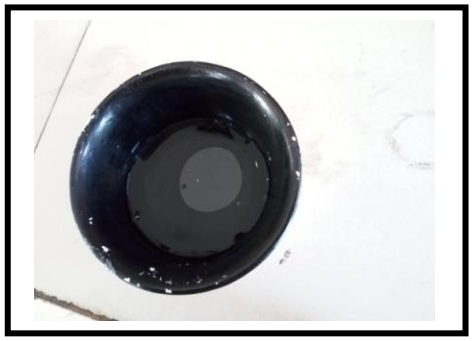
CASE 27

Neikkuri Day- 2

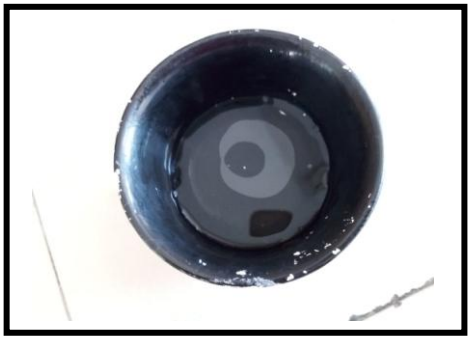
At the moment



I Minute



III Minute



X Minute

Neerkkuri

INTERPRETATION

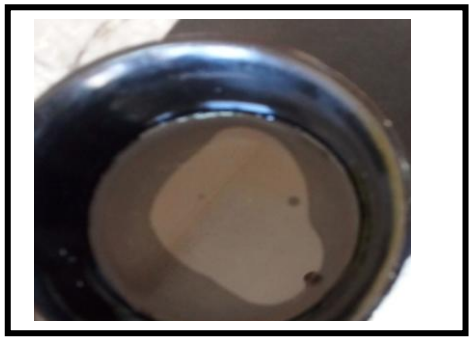
Patient was a known case of Diabetes Mellitus and on urine examination micro Albuminuria and on blood investigation elevated creatinine were shown, Neikkuri examination showed pithathilpitham pattern on second day.

CASE 27

Neikkuri Day 3

At the moment

I Minute



III Minute

X Minute

Neerkkuri

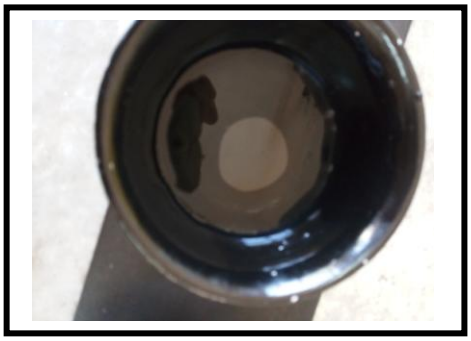
INTERPRETATION

Patient was a known case of Diabetes Mellitus and on urine examination micro Albuminuria and on blood investigation elevated creatinine were shown, Neikkuri examination showed pithathilpitham pattern on first day.

CASE 28

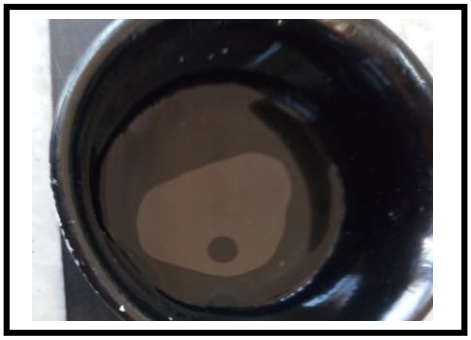
Neikkuri Day- 1

At the moment

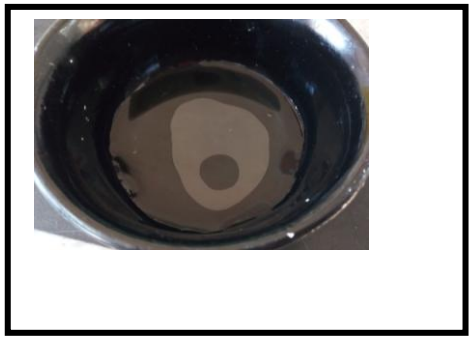


I Minute

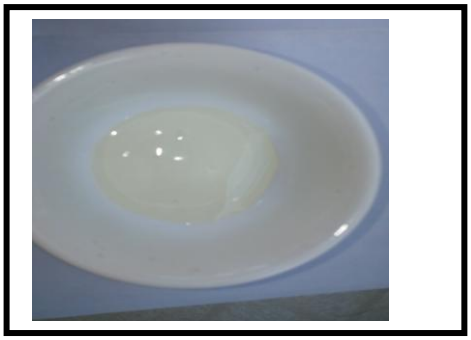
III Minute



X Minute



Neerkkuri



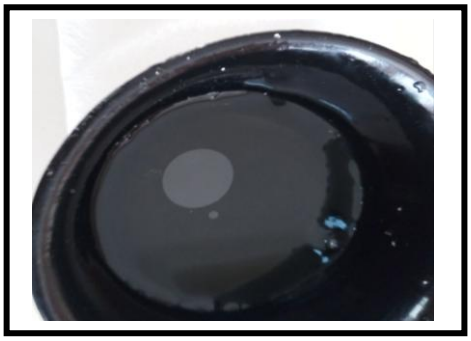
INTERPRETATION

Patient was a known case of Diabetes Mellitus and on urine examination micro Albuminuria was shown, Neikkuri examination showed pithathilpitham pattern on first day.

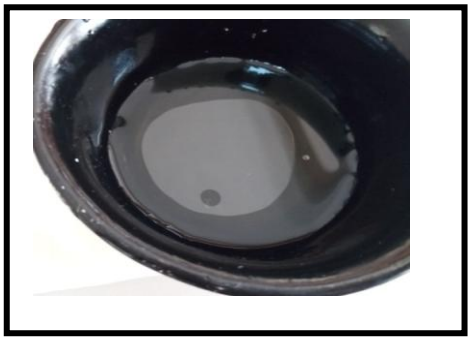
CASE 28

Neikkuri Day 2

At the moment



I Minute



III Minute

X Minute

Neerkkuri

INTERPRETATION

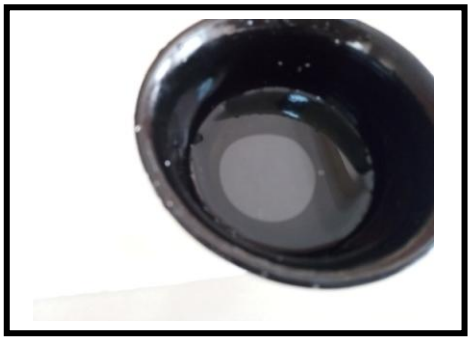
Patient was a known case of Diabetes Mellitus and on urine examination micro Albuminuria was shown, Neikkuri examination showed pithathilpitham pattern on second day.

CASE 28

Neikkuri Day -3

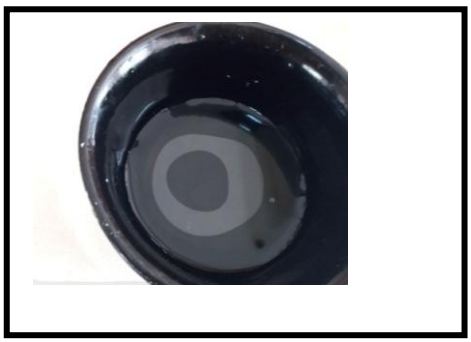
At the moment

I Minute



III Minute

X Minute



Neerkkuri

INTERPRETATION

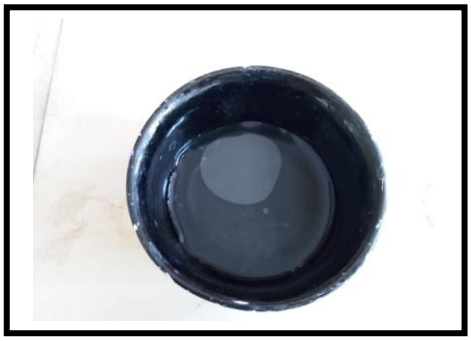
Patient was a known case of Diabetes Mellitus and on urine examination micro Albuminuria was shown, Neikkuri examination showed pithathilpitham pattern on third day.

CASE 29

Neikkuri Day 1

At the moment

I Minute



III Minute

X Minute

Neerkkuri

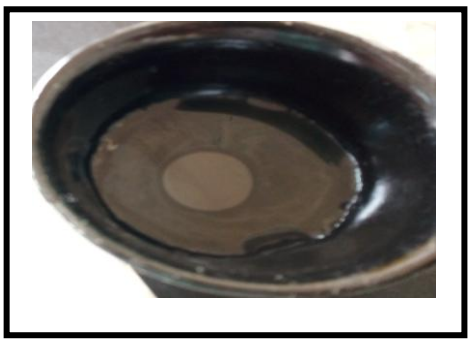
INTERPRETATION

Patient was a known case of Diabetes Mellitus and on urine examination micro Albuminuria was shown, Neikkuri examination showed pitham pattern on first day.

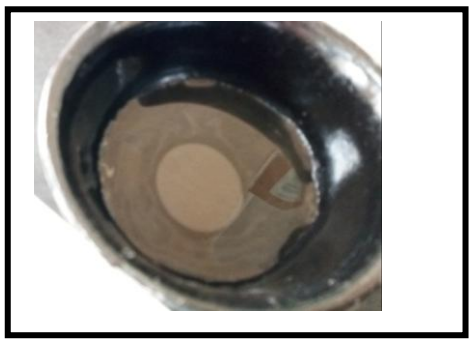
CASE 29

Neikkuri Day 2

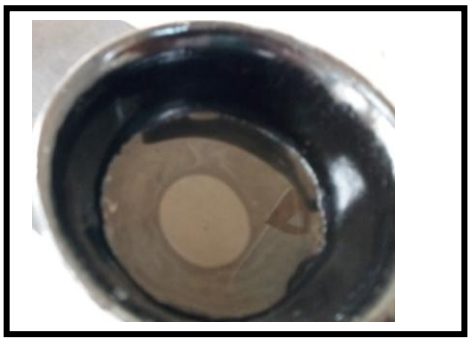
At the moment



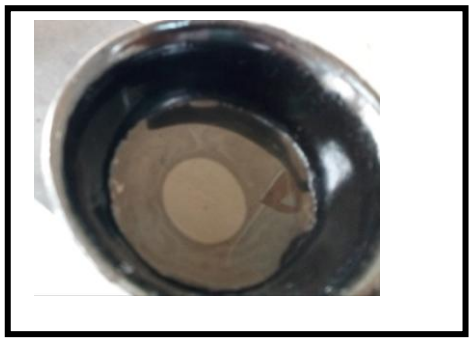
I Minute



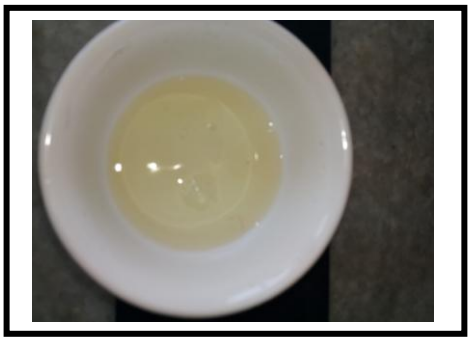
III Minute



X Minute



Neerkkuri



INTERPRETATION

Patient was a known case of Diabetes Mellitus and on urine examination micro Albuminuria was shown, Neikkuri examination showed pithathilpitham pattern on second day.

CASE 29

Neikkuri Day 3

At the moment

I Minute

III Minute

X Minute

Neerkkuri

INTERPRETATION

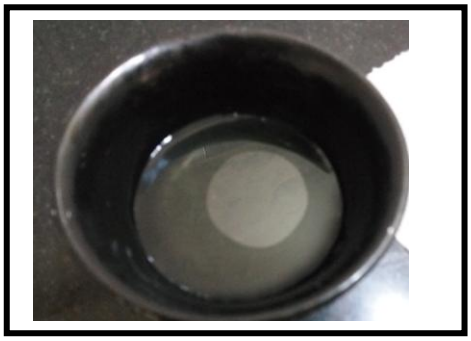
Patient was a known case of Diabetes Mellitus and on urine examination micro Albuminuria was shown, Neikkuri examination showed pithathilpitham pattern on third day.

CASE 30

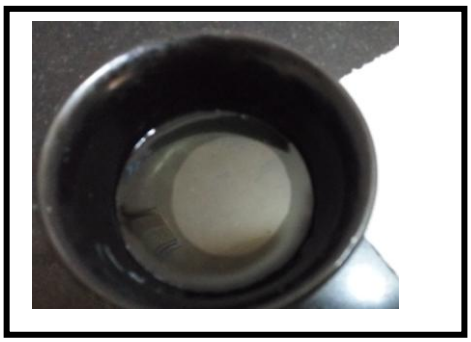
Neikkuri Day 1

At the moment

I Minute



III Minute



X Minute

Neerkkuri

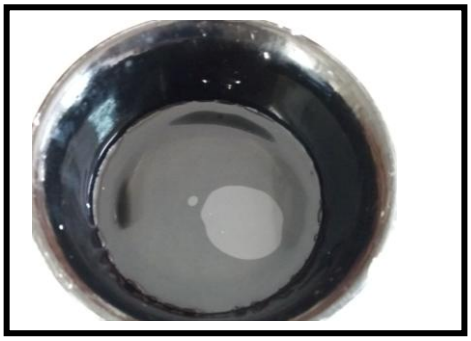
INTERPRETATION

Patient was a known case of Diabetes Mellitus and on urine examination micro Albuminuria was shown, Neikkuri examination showed pitham pattern on first day.

CASE 30

Neikkuri Day 2

At the moment



I Minute

III Minute

X Minute

Neerkkuri

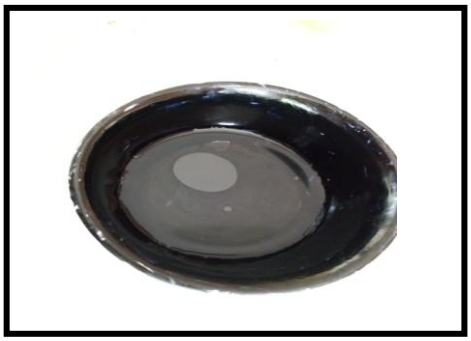
INTERPRETATION

Patient was a known case of Diabetes Mellitus and on urine examination micro Albuminuria was shown, Neikkuri examination showed pitham pattern on second day

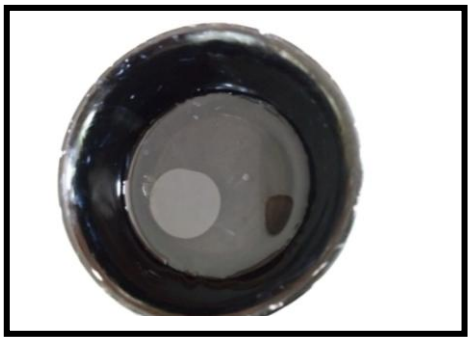
CASE 30

Neikkuri Day 3

At the moment



I Minute



III Minute

X Minute

Neerkkuri

INTERPRETATION

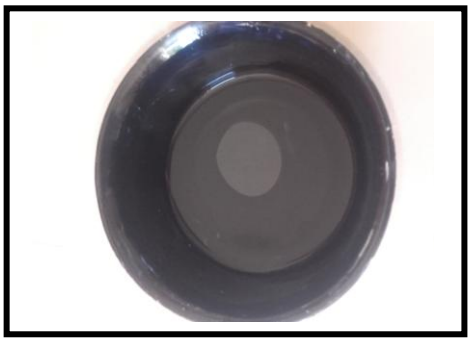
Patient was a known case of Diabetes Mellitus and on urine examination micro Albuminuria was shown, Neikkuri examination showed pitham pattern on first day

Neikkuri images of normal subject

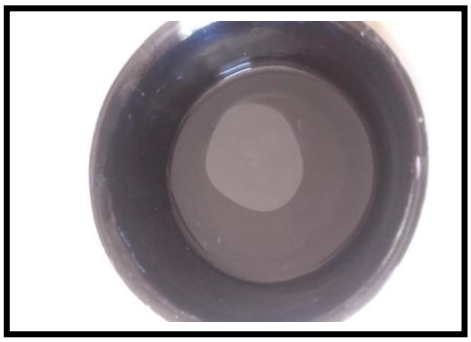
At the moment

I Minute

III Minute



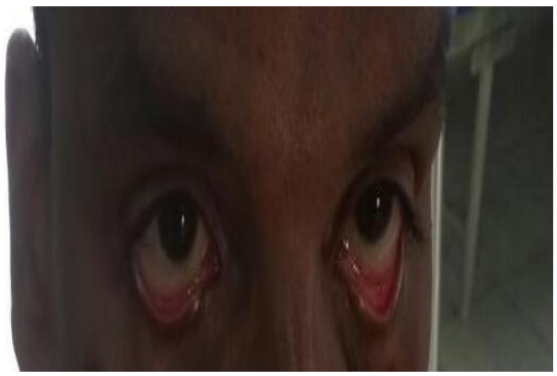
X Minute



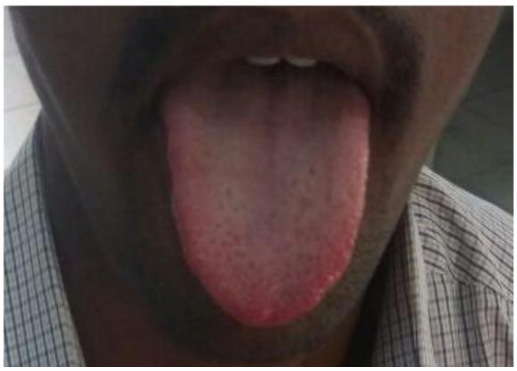
Neerkkuri

VIZHI & NAA EXAMINATION

Normal eye



Normal tongue



Pallor eye

Coated tongue

Black spotted tongue

NATIONAL INSTITUTE OF SIDDHA- राष्ट्रीय सिद्ध संसथान
Ministry of AYUSH- आयुष मंत्रालय
GOVERNMENT OF INDIA-भारत सरकार
TAMBARAM SANATORIUM, CHENNAI -600 047 -ताम्बरम सनटोरियमचेन्नई -600 047
फ़ोन\Tele : 044-22411611 फैक्स\Fax : 22381314
ईमेल: nischennaisiddha@yahoo.co.in वेब :www.nischennai.org

F.No.NIS/6-20/IEC/15-16 Dt: 14.10.2016

CERTIFICATE

Address of Ethics Committee: National Institute of Siddha, Tambaram Sanatorium, Chennai-600047, Tamil Nadu, India	
Principal Investigator: Dr. R.Gayatri– I year, Dept.of Noi Naadal	
Protocol Title:- Neikkuri examination in Athinurai Neer/Frothy Urine-A condition of Albuminuria.	
Documents filed	1) Protocol, 2) Data Collection forms
Clinical trial Protocol (others – Specify)	**Yes-(M.D-Dissertation)**
Informed consent documents	**Yes**
Any other documents	**-**
Date of IEC approval & its number	**NIS/IEC/2016/11-27/ 14.10.2016**

We approve the trial to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study.

(Dr.V.Subramanian)
Chairman

NATIONAL INSTITUTE OF SIDDHA ★ CHENNAI 600 047

(Prof.Dr.V.Banumathi)
Member Secretary

The Tamil Nadu Dr.M.G.R.Medical University

69, Anna Salai, Guindy, Chennai - 600 032.

This Certificate is awarded to Dr/Mr/Mrs.....GAYATRI.R.....

For participating as ~~Resource Person~~ / Delegate in the Twenty First Workshop on

"RESEARCH METHODOLOGY & BIOSTATISTICS"

*For **AYUSH Post Graduates & Researchers***

Organized by the Department of Siddha

The Tamil Nadu Dr. M.G.R. Medical University From 25th to 29th April 2016.

Dr.N.KABILAN, MD(S),
PROF & HEAD
DEPT.OF SIDDHA

Prof.**Dr.P.ARUMUGAM,** M.D.,
REGISTRAR i/c

Prof. **Dr.S.GEETHALAKSHMI,** M.D., Ph.D.,
VICE CHANCELLOR

Clinical Trial Details (PDF Generation Date :- Wed, 20 Jun 2018 09:28:27 GMT)

CTRI Number	CTRI/2017/03/008176 [Registered on: 22/03/2017] - **Trial Registered Prospectively**
Last Modified On	22/03/2017
Post Graduate Thesis	No
Type of Trial	Observational
Type of Study	Case Control Study
Study Design	Single Arm Trial
Public Title of Study	Oil drop test (Neikkuri examination) in Athinurai Neer/Frothy Urine-A condition of Albuminuria
Scientific Title of Study	Neikkuri examination in Athinurai Neer/Frothy Urine-A condition of Albuminuria

Secondary IDs if Any

Secondary ID	Identifier
NIL	NIL

Details of Principal Investigator or overall Trial Coordinator (multi-center study)

Details of Principal Investigator	
Name	GayatriR
Designation	PG Scholar
Affiliation	National Institute of Siddha
Address	Department of noinaadal, National Institute of Siddha, Tambaram sanatorium, Chennai- 600047 Department of noinaadal, National Institute of Siddha, Tambaram sanatorium, Chennai- 600047 Kancheepuram Department of noinaadal, National Institute of Siddha, Tambaram sanatorium, Chennai- 600047 Department of noinaadal, National Institute of Siddha, Tambaram sanatorium, Chennai- 600047 Kancheepuram Kancheepuram TAMIL NADU 600047 India
Phone	9495347599
Fax	
Email	poppysmart126@gmail.com

Details Contact Person (Scientific Query)

Details Contact Person (Scientific Query)	
Name	DR G J Christian
Designation	Associate professor
Affiliation	National Institute of Siddha
Address	Department of noinaadal, National Institute of Siddha, Tambaram sanatorium, Chennai- 600047 Department of noinaadal, National Institute of Siddha, Tambaram sanatorium, Chennai- 600047 Kancheepuram Department of noinaadal, National Institute of Siddha, Tambaram sanatorium, Chennai- 600047 Department of noinaadal, National Institute of Siddha, Tambaram sanatorium, Chennai- 600047 Kancheepuram TAMIL NADU 600047 India
Phone	9962545930
Fax	
Email	christianvijila@gmail.com

Details Contact Person (Public Query)

Details Contact Person (Public Query)	
Name	DR G J Christian

Designation	Associate professor
Affiliation	National Institute of Siddha
Address	Department of noinaadal, National Institute of Siddha, Tambaram sanatorium, Chennai- 600047 Department of noinaadal, National Institute of Siddha, Tambaram sanatorium, Chennai- 600047 Department of noinaadal, National Institute of Siddha, Tambaram sanatorium, Chennai- 600047 Department of noinaadal, National Institute of Siddha, Tambaram sanatorium, Chennai- 600047 Kancheepuram TAMIL NADU 600047 India
Phone	9495347599
Fax	
Email	christianvijila@gmail.com

Source of Monetary or Material Support

Source of Monetary or Material Support
> Ayothidoss pandithar hospital

Primary Sponsor

Primary Sponsor Details	
Name	Ayothidoss pandithar hospital
Address	National institute of siddha, Tambaram sanitorium, Chennai-600047
Type of Sponsor	Research institution and hospital

Details of Secondary Sponsor

Name	Address
NIL	NIL

Countries of Recruitment

List of Countries
India

Sites of Study

Name of Principal Investigator	Name of Site	Site Address	Phone/Fax/Email
Dr Gayatri R	Ayothidoss pandithar hospital	Ayothiudoss pandithar hospital National institute of siddha Tambaram sanatorium Kancheepuram Kancheepuram TAMIL NADU	9495347599 poppysmart126@gmail.com

Details of Ethics Committee

Name of Committee	Approval Status	Date of Approval	Is Independent Ethics Committee?
Institutional ethics committee	Approved	14/10/2016	No

Regulatory Clearance Status from DCGI

Status	Date
Not Applicable	No Date Specified

Health Condition / Problems Studied

Health Type	Condition
Patients	Patients having the complaints of athinurai neer /Frothy urine -A condition of Albuminuria

Intervention / Comparator Agent

Type	Name	Details

Inclusion Criteria

Inclusion Criteria	
Age From	18.00 Day(s)
Age To	70.00 Day(s)
Gender	Both
Details	Patients who are having urine albumin positive with increased froth

	in different examinations

Exclusion Criteria

Exclusion Criteria	
Details	Patients who are undergoing dialysis Patients with high fever Patients with serious systemic illness

Method of Generating Random Sequence	Not Applicable
Method of Concealment	Not Applicable
Blinding/Masking	Not Applicable

Primary Outcome

Outcome	Timepoints
Documentation of various patterns of oil drop shapes in conditions causing increased froth in urine Arriving at a conclusion about specific neikkuri patterns if any conditions causing athinurai neer which may serve as a clue in diagnosis or prognosis Categorisation of disease conditions which cause athinurai neer in humoral basis	Documentation of various patterns of oil drop shapes in conditions causing increased froth in urine Arriving at a conclusion about specific neikkuri patterns if any conditions causing athinurai neer which may serve as a clue in diagnosis or prognosis Categorisation of disease conditions which cause athinurai neer in humoral basis

Secondary Outcome

Outcome	Timepoints
Documentation of various patterns of oil drop shapes in conditions causing increased froth in urine Arriving at a conclusion about specific neikkuri patterns if any conditions causing athinurai neer which may serve as a clue in diagnosis or prognosis Categorisation of disease conditions which cause athinurai neer in humoral basis	Documentation of various patterns of oil drop shapes in conditions causing increased froth in urine Arriving at a conclusion about specific neikkuri patterns if any conditions causing athinurai neer which may serve as a clue in diagnosis or prognosis Categorisation of disease conditions which cause athinurai neer in humoral basis

Target Sample Size	**Total Sample Size=**60 **Sample Size from India=**60
Phase of Trial	N/A
Date of First Enrollment (India)	20/04/2017
Date of First Enrollment (Global)	No Date Specified
Estimated Duration of Trial	**Years=**1 **Months=**0 **Days=**0
Recruitment Status of Trial (Global)	Not Applicable
Recruitment Status of Trial (India)	Not Yet Recruiting
Publication Details	Nil
Brief Summary	Neikkuri is one among the eight kind of diagnostic as well as prognostic technique in siddha .It is cost effective and reliable technique so it would be useful to do documentation of neikkuri in athinurai neer frothy urine A condition of Albuminuria which requires more expensive immuno assay to assess it's progress according to modern system of medicine

E-mail : info@prcivf.com / prashanthhospital2005@gmail.com Web : www.prcivf.com

Name	:MR NARAYANSWAMY R	SIN	:1002856491
OP NO	:201711174067 UHID :10150522	Age / Sex	:70 Years / Male
Sample Date	:17/11/2017 08:46:17	Report Date	:17/11/2017 14:42:32
Referred By	:Dr. Gopalaswamy MS.,FRCS		

Investigation Prescribed By :DR. GOPALASWAMY MS.,FRCS

BIOCHEMISTRY REPORT

Test Name	Result	Units	Reference Range
Plasma Glucose (F)	121	mgs/dl	70 - 110
Plasma Glucose (2 Hrs. PP)	140	mgs/dl	upto 140
Creatinine(serum)	0.9	mgs/dl	0.6 - 1.2
Lipid Profile			
Total Cholestrol	204 [H]	mgs/dl	150 - 200
Triglycerides	73	mgs/dl	UPTO : 150 - Normal 150 - 199 Border Line high 200 - 499 - High
HDL Cholesterol (Direct)	37	mgs/dl	35 - 55
LDL (Direct)	101	mgs/dl	Less than 130
VLDL	15	mgs/dl	< 35
Total HDL Ratio	5.5	mgs/dl	3.5 - 5.5
HbA1c	6.9	%	Non Diabetic : <6.0 Good Control : 6.0 - 7.0 Fair Control : 7.0 - 8.0 Poor Control : >8.0
Microalbuminuria	368.0	mg/l	upto 15

Mrs. J. JANAKI, DMLT
Senior Lab Technician
Checked By
Kindly Co-relate values with clinical findings

Seen By Doctor

Signature
Mr. S. SATHISH, DML
Lab Supervisor

This report is electronically generated

LABORATORY REPORTS NEED CORRELATION WITH CLINICAL EXAMINATION AND RESULTS OF OTHER TESTS TO ARRIVE AT THE DIAGNOSIS

HITECH DIAGNOSTIC CENTRE
The Extra Care Lab
No.935, GKS Tower, Poonamallee High Road, Purasawalkam, Chennai - 600 084

Patient :P0433096 **Mr. RAVICHANDRAN.K (64/M)**
SID.No. :**004305**
Branch :**SALIGRAMAM BRANCH**
Referrer :**Dr.GAYATHRI..**
Ph :9445755808

Date :31/01/2018
Rec Time :07:24:59
Rpt Date :31/01/2018
Rpt Time :16:33:54
Page # :1 / 1
Final Report

Test	Result	Biological Reference Interval

TEST REPORT

URINE - BIOCHEMISTRY

MICROALBUMIN - SPOT

Specimen : URINE
Method : Immunoturbidimetry
Spot Microalbumin : 4130.7 ug/mg of creatinine

Ref.Value :
Normal : Less than 30 ug/mg of creatinine
Microalbumiuria : 30 - 299 ug/mg of creatinine
Clinical Albuminuria : More than 300 ug/mg of creatinine

F & PP GLUCOSE PROFILE (2 HOURS)

BLOOD - BIOCHEMISTRY

GLUCOSE (FASTING) : 78 mg/dl

Specimen : FLUORIDE PLASMA
Method : Hexokinase

74 - 99 mg/dl : Normal.
100 - 110 mg/dl: IFG/Good Controle
110 - 126 mg/dl: IFG/Fair Controle
> 126 mg/dl : DM / Poor Controle

IFG- Impaired Fasting Glucose. Pls do GTT to confirm Diagnosis.

GLUCOSE (PP 2 HOURS) : 152 mg/dl

Upto 140 mg/dl

Specimen : FLUORIDE PLASMA
Method : Hexokinase

CLINICAL PATHOLOGY

SUGAR (FASTING)(GLU) - URINE : Nil

Specimen : URINE
Method : DIPSTICK

SUGAR (2 HOURS)(GLU) - URINE : Nil

Specimen : URINE
Method : DIPSTICK

EPORT

Name : Mrs.PRABHAVATHY

Age/Sex : 73 Years / Female

Ref.By : DR.

Sample No : 31515

SID Date : 25/09/20

Rpt Date : 25/09/20

FINAL TEST REPORT

TEST NAME	RESULTS/UNITS	REFERENCE VALUE
BIOCHEMISTRY		
Blood UREA	: 42.0 mg/dl	13 - 45 mg/dl
Method : (UREASE/GLDH)		
SERUM CREATININE	: 1.4 mg/dl	Mele: 0.6 - 1.5
Method : (JAFFES)		Female:0.6 - 1.4
24 Hrs Urine Protein		
Total Volume	: 1250 ml/24hrs	
Urine Protein	: 104.5 mg/dl	
Protein 24 hr	: 1306.2 mg/day	Adult : <100
Method : (Colorimetric : Pyrogallol red)		Pregnancy : <150

End of Report

S. Rajesh

S.Rajesh

Sr.Lab Manager

LAB TECHNOLOGIST

Refer to conditions of reporting overleaf ***Referred Test* *Results relate only to the sample as received*

The Laboratory is a regular participant of Internal and External Assurance Programmes with Randox (UK) & CMC (Vellore)

Nathan's Clinical Service

L.A.B, E.C.G, & X-RAY

e – mail ID : ulaganathanlabs@gmail.com, Whatsapp: 944 5111 991

No. 13, Nehru Street, Chitlapakkam, Ch –64(Back Side to Varadharaja Theater)

Name : **Mr. VELMURUGAN**
Age : 65 Years
Sex : Male

Ref. By : **NATHANS CLINICAL SERVICE.**

SID No : VELC 002626
Registered Date : 06/02/2018 / 13:55
Collected Date : 06/02/2018
Reported Date : 06/02/2018 / 18:55

Sample Collected and sent

FINAL TEST REPORT

TEST DESCRIPTION	RESULT	UNIT	BIOLOGICAL REFERENCE INTERVAL
MICROALBUMINUREA (Urine/Immunoturbidimetric)	213.4	mg/L	UP TO 20

End of the Report

DR. KOMALA.G. MD.,
CONSULTANT BIOCHEMIST

DR. KOMALA.G. MD.,
CONSULTANT BIOCHEMIST

Special Packages: Wellness Package, Bone Profile, Vitamin's Package, Diabetic Monitor Arthritis Profile, Liver Functions, Kidney Functions, Thyroid's Functions, Pre-Pregnancy, Pregnancy and Hair Loss Profile's

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GG Gee. Gee. LAB
New No. 75/1, Karaneeswarar Koil Street, Mylapore,
Chennai - 600 004, Ph : 2498 6196 / Cell : 98419 44164
Email : geegeelab1972@gmail.com

Name : **MRS. PARIMALA**
Age/Sex : 40 Years / Female
Ref By : **DR.R.KEDARNATHAN.,MD.,**

Reg.No.: 29368
S.ID. : 29368
Date : 13/03/2018

LABORATORY REPORT

Test Name	Result/Units	Reference Value
BIOCHEMISTRY		
Blood Sugar (F) Method : GOD/POD	: 215 mg/dL	70 - 110 mg/dl
Blood Sugar (PP) 2 Hrs Method : GOD/POD	: 276 mg/dl	90 - 140 mg/dl
Blood Urea	: 36 mg/dL	15 - 40
Serum Creatinine	: 2.0 mg/dL	Male :0.4 - 1.4 Female :0.4 - 1.2
URINE BIOCHEMISTRY		
Spot Microalbumin	: 456 mg / l	Less than 30.0 mg/ l
CLINICAL PATHOLOGY		
URINE ROUTINE EXAMINATION		
Colour	: Straw	
Transparency	: S.Turbid	
Albumin	: (++)	
Sugar	: (+)	
Bile salt	: Nil	
Bile Pigment	: Nil	
DEPOSITS		
Pus Cells	: 01 - 02/hpf	
Epithelial Cells	: 01 - 02/hpf	
RBCs	: Nil	
Cast	: Nil	
Crystals	: Nil	

End of Report

Received Date : 13/03/2018
Reported Date : 13/03/2018

LAB INCHARGE

VRR Diagnostics

Reliable always

Branch :MADIPAKKAM
Patient :0200166313 Mrs. SARASWATHY U (59 Y/F)
SID.No. :02054225
Referrer :Dr. GAYATHRI

SID Date :23/12/2017
Reg Time :12:11:09
Rpt Date :23/12/2017
Rpt Time :16:13:32

Test	Result	Biological Reference Interval
SPOT URINE ANALYSIS		
MICROALBUMINURIA (SU) Method : Immunoturbidimetry Specimen : Urine	*276.0 mg/L	< 30

Comments :Rechecked

*** End of the Report ***

Verified by

DR.P. RASAPPAN Ph.D.,D.Sc
DIRECTOR

Patient	: MR. RAJU.J	SID No.	: 26014702
Age /Sex	: 63 Y / Male	Reg.Date & Time	: 06/05/2018 12:50
Referrer	: SAI HS DIAGNOSTIC CENTER	Coll Date & Time	: 06/05/2018 13:51
Branch	: VADAPALANI-LAB	Report Date & Time	: 06/05/2018 18:20

INVESTIGATION / METHOD	RESULT	UNITS	BIOLOGICAL REFERENCE INTERVAL
BIOCHEMISTRY			
MICROALBUMIN - SPOT URINE(ACR RATIO)(Nephelometry)			
MICROALBUMIN-SPOT URINE	**1847***	mg/L	<20
CREATININE (SPOT URINE)	96.9	mg/dl	40-300
MICROALBUMIN/CREATININE RATIO(ACR Ratio)	1906*	ug/mg of creat	< 30

Comments :* Rechecked kindly correlate clinically
Confirmation of persistent microalbuminuria within 3 months of initial detection is recommended by the American Diabetes Association and the National Kidney Foundation.

End of the Report

DR.S.ASOKKUMAR, M.Sc.,Ph.D.,
Biochemist

06/05/2018.
L.RAMESH
9940392307
SAI HS DIAGNOSTIC CENTRE
No.41, Gandhi Road,
Perungalathur, Chennai-63.

HITECH DIAGNOSTIC CENTRE

The Extra Care Lab

No.935, GKS Tower, Poonamallee High Road, Purasawalkam, Chennai - 600 084

An ISO 9001:2015 Certified Organisation

Patient : P0563264 **Mr. ASHOK KUMAR (39/M)**

SID.No. : **024108**

Branch : **SALIGRAMAM BRANCH**

Referrer : **Dr.SELF**

Ph :9444802607

Date :08/05/2017

Rec Time :08:31:17

Rpt Date :08/05/2017

Rpt Time :14:37:38

Page # :1 / 1

Final Report

TEST REPORT

Test	Result	Biological Reference Interval
URINE - BIOCHEMISTRY		
24 HOURS URINE PROTEINS	: 1569.0 mg/24 Hrs	At rest : UPTO 100 mg/24 Hrs Pregnancy : UPTO 150 mg/24 Hrs After Exercise: upto 300 mg/24 hrs
Specimen : URINE		
Method : Turbidimetric		

Ganesan

DR.SP.GANESAN. MBBS.,DCP.,

* End Of Report *

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DEPARTMENT OF NOI NAADAL

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47.

NEIKKURI EXAMINATION IN ATHI NURAI NEER /FROTHY URINE

A CONDITION OF ALBUMINURIA

FORM I - SCREENING AND SELECTION PROFORMA

1. O.P.No _____ 2. I.P No _____ 3. Bed No: _____ 4. S.No: _____

5. Name: _____6. Age (years): ☐ 7. Gender: M ☐ ☐

8. Occupation: _____ 9. Income: _____

10. Address: .....

.....

.....

11. Contact Nos: ----------

12. E-mail : ----------

13. Whether taken any other medication for the same disease previously YES ☐ NO ☐

If yes,
Name of the medicines :

Duration :

If resorted to Siddha medicine for the treatment of YES ☐ NO ☐

Athi Nurai Neer

Reasons for resorting to Siddha medicine :

	YES	NO
(a) Cost effectiveness :	☐	☐
(b) No side effects in Siddha medicine :	☐	☐
(c) Dissatisfaction with the previous treatment :	☐	☐

INCLUSION CRITERIA

	YES	NO
1. Age 25-70	☐	☐
2. Patients who are having urine albumin positive in different examinations and increased froth in urine	☐	☐

EXCLUSION CRITERIA

	YES	NO
• Patients who are undergoing dialysis	☐	☐
• Patients with high fever	☐	☐
• Patients with serious systemic illness	☐	☐

Date : **P.G Student** **Lecturer**

DEPARTMENT OF NOI NAADAL

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47.

NEIKKURI EXAMINATION IN ATHI NURAI NEER /FROTHY URINE

A CONDITION OF ALBUMINURIA

FORM I A - HISTORY PROFORMA

1. Sl.No of the case: _____

2. Name: _____ Height: _____ cms Weight: _____ Kg

3. Age (years): _____ DOB ☐☐ ☐☐ ☐☐☐☐
D D M M Y E A R

4. Educational Status:

1) Illiterate ☐ 2) Literate ☐ 3) Student ☐ 4) Graduate/Postgraduate ☐

5. Nature of work:

1) Sedentary work ☐

2) Field work with physical labour ☐

3) Field work Executive ☐

6. Complaints and Duration:

_____

_____

7. History of present illness:

_____

_____

_____

8. History of Past illness:

	1. Yes	2. No
Systemic hypertension	☐	☐
Ischemic heart disease	☐	☐
Dyslipidaemia	☐	☐

Jaundice	☐	☐
Bronchial asthma	☐	☐
Any drug allergy	☐	☐
Any surgeries	☐	☐
Any major illnesses	☐	☐

9.Habits:

	1. Yes	2. No
Smoker	☐	☐
Alcoholic	☐	☐
Drug Addiction	☐	☐
Betel nut chewer:	☐	☐
Tea	☐	☐
Coffee	☐	☐
Milk	☐	☐

DIET HISTORY

Type of diet ☐ V NV ☐ M ☐

VEGETARIAN FOODS	1. Yes	2. No
sweets	☐	☐
Ice creams	☐	☐
Junk foods	☐	☐

NON VEGETARIAN FOODS

Meat	☐	☐
Fish	☐	☐
Crab	☐	☐

DRINKS

Soft drinks	☐	☐

10. Personal history:

Marital status: Married ☐ Unmarried ☐

No. of children: Male: _____ Female: _____

Socio economic status:

11. Family history:

13. Others:

Date : **P.G Student** **sLecturer**

DEPARTMENT OF NOI NAADAL

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47.

NEIKKURI EXAMINATION IN ATHI NURAI NEER /FROTHY URINE

A CONDITION OF ALBUMINURIA

FORM II -CLINICAL ASSESSMENT

1. Serial No: ________

2. Name: ________________

3. Date of birth: [ ][ ] [ ][ ] [ ][ ][ ][ ]
D D MM YEAR

4. Age: _______ years

5. Date: ___________

GENERAL EXAMINATION:

1. Height: ________ cms. BMI ______ (Weight Kg/ Height m2)

2. Weight (kg):

3. Temperature (°F):

4. Pulse rate:

5. Heart rate:

6. Respiratory rate:

7. Blood pressure:

8. Pallor:

9. Jaundice:

10. Cyanosis:

11. Lymphadenopathy:

12. Pedal edema:

13. Clubbing:
14. Jugular vein pulsation

EXAMINATION

1.Inspection

2.palpation

3..Percussion

4.auscultation

VITAL ORGANS EXAMINATION

	1. Normal	2. Affected
1. Heart	☐	☐ _____
2. Lungs	☐	☐ _____
3. Brain	☐	☐ _____
4. Liver	☐	☐ _____
5. Kidney	☐	☐ _____
6. Spleen	☐	☐ _____
7. Stomach	☐	☐ _____

SYSTEMIC EXAMINATION:

1. Cardio Vascular System _____
2. Respiratory System _____
3. Gastrointestinal System _____
4. Central Nervous System _____
5. Uro genital System _____
6. Endocrine System _____

SIDDHA SYSTEM OF EXAMINATION

[1] ENVAGAI THERVU [EIGHT-FOLD EXAMINATION]

I. NAADI (KAI KURI) (RADIAL PULSE READING)

(a) Naadi Nithanam (Pulse Appraisal)

1. Kalam (Pulse reading season)

1. Kaarkaalam (Rainy season)	☐	2.Koothirkaalam (Autumn)	☐
3. Munpanikaalam (Early winter)	☐	4.Pinpanikaalam (Late winter)	☐
5. Ilavenirkaalam (Early summer)	☐	6.Muthuvenirkaalam (Late summer)	☐

2. Desam (Climate of the patient's habitat)

1. Kulir (Temperate)	☐	2. Veppam (Hot)	☐

3. Vayathu (Age)1. 1-33yrs	☐	2. 34-66yrs	☐	3. 67-100	☐

4. Udal Vanmai (General body condition)

1. Iyyalbu (Normal built)	☐	3. Valivu (Robust)	☐	4.Melivu (Lean)	☐

5. Vanmai (Expansile Nature)

1. Vanmai	☐	2.Menmai	☐

6. Panbu (Habit)

1. Thannadai (Playing in)	☐	2. Puranadai (Playing out)	☐	3.Illaitthal (Feeble)	☐
4. Kathithal (Swelling)	☐	5.Kuthithal (Jumping)	☐	6.Thullal (Frisking)	☐

7. Azhutthal (Ducking)	☐	8. Padutthal (Lying)	☐	9. Kalatthal (Blending)	☐
10. Munnokku (Advancing)	☐	11. Pinnokku (Flinching)	☐	12. Suzhalal (Revolving)	☐
13. Pakkamnokku (Swerving)	☐				

(b) Naadi nadai (Pulse Play)

1. Vali	☐	2. Azhal	☐	3. Iyyam	☐
4. Vali Azhal	☐	5. Azhal Vali	☐	6. Iyya Vali	☐
7. Vali Iyyam	☐	8. Azhal Iyyam	☐	9. Iyya Azhal	☐

II.NAA (TONGUE)

1. Maa Padinthiruthal (Coatedness)	1. Present	☐	2. Absent	☐		
2. Niram (Colour)	1.Karuppu (Dark)	☐	2. Manjal (Yellow)	☐	3. Velluppu (Pale)	☐
3. Suvai (Taste sensation)	1.Pulippu (Sour)	☐	2. Kaippu (Bitter)	☐	3. Inippu (Sweet)	☐
4. Vedippu (Fissure)	1. Absent	☐	2. Present	☐		
5. Vai neer ooral (Salivation)	1.Normal	☐	2. Increased	☐	3.Reduced	☐

III.NIRAM (COMPLEXION)

1. Karuppu (Dark)	☐	2.Manjal (Yellowish)	☐	3.Velluppu (Fair)	☐

IV. MOZHI (VOICE)

1. Sama oli (Medium pitched)	☐	Urattha oli (High pitched)	☐	3.Thazhantha oli (Low pitched)	☐

V. VIZHI (EYES)

1. Niram (Venvizhi)
(Discolouration)

1. Karuppu (Dark) ☐ 2. Manjal (Yellow) ☐

3. Sivappu (Red) ☐ 4.Velluppu (White) ☐

5. No Discoloration ☐

2. Kanneer (Tears) 1.Normal ☐ 2. Increased ☐ 3.Reduced ☐

3. Erichchal (Burning sensation) 1.Present ☐ Absent ☐

4. Peelai seruthal (Mucus excrements) 1.Present ☐ Absent ☐

VI. MEI KURI (PHYSICAL SIGNS)

1. Veppam (Warmth) 1. Mitham (Mild) ☐ 2. Migu (Moderate) ☐ 3. Thatpam (Low) ☐

2. Viyarvai (Sweat) 1. Increased ☐ 2. Normal ☐ 3. Reduced ☐

3. Thodu vali (Tenderness) 1. Absent ☐ 2. Present ☐

VII. MALAM (STOOLS)

1. Niram (Color) 1. Karuppu (Dark) ☐ 2. Manjal (Yellowish) ☐

3. Sivappu (Reddish) ☐ 4. Velluppu (Pale) ☐

2. Sikkal (Constipation) 1. Present ☐ 2. Absent ☐

3. Sirutthal (Poorly formed stools) 1. Present ☐ 2. Absent ☐

4. Kalichchal (Loose watery stools) 1. Presen ☐ 2. Absent ☐

5. Seetham (Watery and mucoid excrements) 1. Present ☐ 2. Absent ☐

6. Vemmai (Warmth) 1. Present ☐ 2. Absent ☐

7. History of habitual constipation 1. Present ☐ 2. Absent ☐

8. Passing of a) Mucous 1. Yes ☐ 2. No ☐

b) Blood 1. Yes ☐ 2. No ☐

VIII. MOOTHIRAM (URINE)

(a) NEER KURI (PHYSICAL CHARACTERISTICS)

1. Niram (colour)

Colourless	☐	Milky purulent	☐	orange	☐
Red	☐	Greenish	☐	dark brown	☐
Bright red	☐	Black	☐	Brown red or yellow	☐

2. Manam (odour)		Yes	No
Ammonical	:	☐	☐
Fruity	:	☐	☐
Others	:	_____	

3. Edai (Specific gravity)		Yes	No
Normal (1.010-1.025)	:	☐	☐
High Specific gravity (>1.025)	:	☐	☐
Low Specific gravity (<1.010)	:	☐	☐
Low and fixed Specific gravity (1.010-1.012):		☐	☐

4. Alavu(volume)		Yes	No
Normal (1.2-1.5 lt/day)	:	☐	☐
Polyuria (>2lt/day)	:	☐	☐
Oliguria (<500ml/day)	:	☐	☐

5. Nurai(froth)		Yes	No
Clear	:	☐	☐
Cloudy	:	☐	☐

6. **Enjal (deposits)** : Yes ☐ ☐

b) NEI KURI (oil spreading sign)

1. Aravam (Serpentine fashion)	☐	2. Mothiram (Ring)	☐
3. Muthu (Pearl beaded appear)	☐	4. Aravil Mothiram (Serpentine in ring fashion)	☐
5. Aravil Muthu (Serpentine and Pearl patterns)	☐	6. Mothirathil Muthu (Ring in pearl fashion)	☐
7. Mothirathil Aravam (Ring in Serpentine fashion)	☐	8. Muthil Aravam (Pearl in Serpentine fashion)	☐
9. Muthil Mothiram (Pearl in ring fashion)	☐	10. Asathiyam (Incurable)	☐
11. Mellena paraval (Slow spreading)	☐		

12. others:_____

[2]. MANIKADAI NOOL (Wrist circummetric sign) : _____ fbs

[3]. **IYMPORIGAL /IYMPULANGAL**

(Penta sensors and its modalities)

	1. Normal	**2. Affected**
1. Mei (skin)	☐	☐
2. Vaai (Mouth/ Tongue)	☐	☐
3. Kan (Eyes)	☐	☐
4. Mookku (Nose)	☐	☐
5. Sevi (Ears)	☐	☐

[4]. KANMENTHIRIYANGAL /KANMAVIDAYANGAL
(Motor machinery and its execution)

	1. Normal	**2. Affected**
1. Kai (Hands)	☐	☐
2. Kaal (Legs)	☐	☐
3. Vaai (Mouth)	☐	☐
4. Eruvai (Analepy)	☐	☐
5. Karuvaai (Birth canal)	☐	☐

[5]. YAKKAI (SOMATIC TYPES)

Vatha constitution	Pitha constitution	Kaba constitution
Lean and lanky built ☐	Thin covering of bones and joints by soft tissue ☐	Plumpy joints and limbs ☐
Hefty proximities of limbs ☐	Always found with warmth, sweating and offensive body odour ☐	Broad forehead and chest ☐
Cracking sound of joints on walking ☐	Wrinkles in the skin ☐	Sparkling eyes with clear sight ☐
Dark and thicker eye lashes ☐	Red and yellow admixed complexion ☐	Lolling walk ☐
Dark and light admixed complexion ☐	Easily suffusing eyes due to heat and alcohol ☐	Immense strength despite poor eating ☐
Split hair ☐	Sparse hair with greying ☐	High tolerance to hunger, thirst and fear ☐
Clear words ☐	Intolerance to hunger, thirst and heat ☐	Exemplary character with good memory power ☐
Scant appetite for cold food items ☐	Inclination towards perfumes like sandal ☐	More liking for sweet taste ☐
Poor strength despite much eating ☐	Slender eye lashes ☐	Husky voice ☐
Loss of libido ☐	Pimples and moles are plenty ☐	
In generosity ☐		
Sleeping with eyes half closed ☐		

RESULTANT SOMATIC TYPE: _____

[6] GUNAM

1. Sathuva Gunam ☐
2. Rajo Gunam ☐
3. Thamo Gunam ☐

[7] UYIR THATHUKKAL

A. VALI

	1. Normal	**2. Affected**
1. Praanan (Heart centre)	☐	☐
2. Abaanan (Matedial of muladhar centre)	☐	☐
3. Samaanan (Navel centre)	☐	☐
4. Udhaanan (Forehead centre)	☐	☐
5. Viyaanan (Throat centre)	☐	☐
6. Naahan (Higher intellectual function)	☐	☐
7. Koorman (Air of yawning)	☐	☐
8. Kirukaran (Air of salivation)	☐	☐
9. Devathathan (Air of laziness)	☐	☐
10. Dhananjeyan (Air that acts on death)	☐	☐

B. AZHAL

	1. Normal	**2. Affected**
1. Anala pittham (Gastric juice)	☐	☐
2. Prasaka pittham (Bile)	☐	☐
3. Ranjaka pittham (Haemoglobin)	☐	☐
4. Aalosaka pittham (Aqueous Humour)	☐	☐
5. Saathaka pittham (Life energy)	☐	☐

C. IYYAM

	1. Normal	**2. Affected**
1. Avalambagam (Serum)	☐	☐
2. Kilethagam (saliva)	☐	☐
3. Pothagam (lymph)	☐	☐
4. Tharpagam (cerebrospinal fluid)	☐	☐
5. Santhigam (Synovial fluid)	☐	☐

[8] UDAL THATHUKKAL

INCREASED SAARAM (CHYLE)		DECREASED SAARAM(CHYLE)	
Loss of appetite	☐	Loss weight	☐
Excessive salivation	☐	Tiredness	☐
Loss of perseverance	☐	Dryness of the skin	☐
Excessive heaviness White musculature	☐	Diminished activity of the sense organs	☐
Cough, dysponea, excessive sleep	☐		
Weakness in all joints of the body	☐		

A. SAARAM: INCREASED ☐ DECREASED ☐

INCREASED CENNEER(BLOOD)		DECREASED CENNEER(BLOOD)	
Boils in different parts of the body	☐	Anemia	☐
Anorexia	☐	Tiredness	☐
Mental disorder	☐	Neuritis	☐
Spleenomegaly	☐	Lassitude	☐
Colic pain	☐	Pallor of the body	☐
Increased pressure	☐		
Reddish eye and skin	☐		
Jaundice	☐		
Haematuria	☐		

B. CENNEER: INCREASED ☐ DECREASED ☐

INCREASED OON (MUSLE)		DECREASED OON (MUSLE)	
Cervical lymphadenitis	☐	Impairment of sense organs	☐
Vernical ulcer	☐	Joint pain	☐
Tumour in face ,abdomen, thigh, genitalia	☐	Jaw, thigh and genitalia gets shortened	☐
Hyper muscular in the cervical region	☐		

C. OON: INCREASED ☐ DECREASED ☐

INCREASED KOZHUPPU (ADIPOSE TISSUE)		DECREASED KOZHUPPU (ADIPOSE TISSUE)	
Cervical lymph adenitis		Pain in the hip region	
Vernical ulcer		Disease of the spleen	
Tumour in face, abdomen, thigh, genitalia			
Hyper muscular in the cervical region			
Dyspnoea			
Loss of activity			

D. KOZHUPPU: INCREASED DECREASED

INCREASED ENBU (BONE)		DECREASED ENBU (BONE)	
Growth in bones and teeth		Bones diseases	
		Loosening of teeth	
		Nails splitting	
		Falling of hair	

E. ENBU: INCREASED DECREASED

INCREASED MOOLAI (BONE MARROW)		DECREASED MOOLAI (BONE MARROW)	
Heaviness of the body		Osteoporosis	
Swollen eyes		Sunken eyes	
Swollen phalanges chubby fingers			
Oliguria			
Non healing ulcer			

F. MOOLAI: INCREASED DECREASED

INCREASED SUKKILAM/SURONITHAM (SPERM OR OVUM)		DECREASED SUKKILAM/SURONITHAM (SPERM OR OVUM)	
Infatuation and lust towards women / men	☐	Failure in reproduction	☐
Urinary calculi	☐	Pain in the genitalia	☐

G. SUKKILAM/SURONITHAM: INCREASED ☐ DECREASED ☐

[9] MUKKUTRA MIGU GUNAM

I. Vali Migu Gunam	**1. Present**	**2. Absent**
1. Emaciation	☐	☐
2. Complexion – blackish	☐	☐
3. Desire to take hot food	☐	☐
4. Shivering of body	☐	☐
5. Abdominal distension	☐	☐
6. Constipation	☐	☐
7. Insomnia	☐	☐
8. Weakness	☐	☐
9. Defect of sense organs	☐	☐
10. Giddiness	☐	☐
11. Lake of interest	☐	☐

I. Pitham Migu Gunam	**1. Present**	**2. Absent**
1. Yellowish discolouration of skin	☐	☐
2. Yellowish discolouration of the eye	☐	☐
3. Yellow coloured urine	☐	☐
4. Yellowishness of faeces	☐	☐
5. Increased appetite	☐	☐
6. Increased thirst	☐	☐
7. Burning sensation over the body	☐	☐
8. Sleep disturbance	☐	☐

III. Kapham migu gunam	**1. Present**	**2. Absent**
1. Increased salivary secretion	☐	☐
2. Reduced activeness	☐	☐
3. Heaviness of the body	☐	☐
4. Body colour – fair complexion	☐	☐
5. Chillness of the body	☐	☐
6. Reduced appetitie .	☐	☐
7. Eraippu	☐	☐
8. Increased sleep	☐	☐

[10]**. NOIUTRA KALAM**

1. Kaarkaalam (Aug15-Oct14)	☐	2.Koothirkaalam (Oct15-Dec14)	☐
3. Munpanikaalam (Dec15-Feb14)	☐	4.Pinpanikaalam (Feb15-Apr14)	☐
5. Ilavanirkaalam (Apr15-June14)	☐	6.Muthuvenirkaalam (June15-Aug14)	☐

[11]**. NOI UTRA NILAM**

1. Kurunji (Hilly terrain) ☐ 2. Mullai (Plains) ☐ 3. Marutham ☐

4. Neithal (Coastal belt) ☐ 5. Paalai (Desert) ☐

[12].Date of Birth ☐☐ ☐☐ ☐☐☐☐

[13]. Time of Birth ☐ AM ☐ PM ☐

[14]. Place of Birth: _____

CLINICAL SYMPTOMS OF "athi nurai neer" :

	YES	NO
1. Increased froth in urine.	☐	☐

Date : **P.G Student** **Lecture**

DEPARTMENT OF NOI NAADAL

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47.

NEIKKURI EXAMINATION IN ATHI NURAI NEER /FROTHY URINE

A CONDITION OF ALBUMINURIA

FORM-III-LABORATORY INVESTIGATIONS

1. O.P No: _____ Lab.No_____ Serial No_____

2. Name: _____

3.Date of birth : | | | | | | | | | |
D D M M Y E A R

4. Age : _____ years

5. Date of assesssment: _____

BLOOD

1. TC _____ Cells/cu mm

2. DC

P___% L _____% E _____% M _____% B_____%

3. Hb _____ gms%

4. ESR At 30 minutes _____ mm At 60 minutes _____mm

5. Blood Sugar-F_____mgs%

6. Blood Sugar-PP _____mg%

7. Serum Cholesterol _____mgs %

8. HDL _____ mgs%

9. LDL _____mgs%

10. Triglycerides _____mgs%

11. Blood Urea _____mgs%

12. Serum Creatinine _____mgs%

URINE

1. Neerkuri _____

2. Neikuri _____

3. Sugar F&PP _____

4. Albumin _____

5. Deposits _____

MOTION

1. Ova
2. Cyst
3. Occult blood

Date : **P.G Student** **Lecturer**

DEPARTMENT OF NOI NAADAL

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47.

NEIKKURI EXAMINATION IN ATHI NURAI NEER /FROTHY URINE

A CONDITION OF ALBUMINURIA

FORM IV - INFORMED WRITTEN CONSENT FORM

I ……..exercising my free power of choice, hereby give my consent to be included as a subject in the diagnostic trial entitled " Neikkuri Examination in Athi Nurai Neer/Frothy Urine –A condition of Albuminuria.. I may be asked to give urine and blood samples during the study

I have been informed about the study to my satisfaction by the attending investigator about the purpose of this trial, the nature of study and the laboratory investigations. I also give my consent to publish my study results in scientific conferences and reputed scientific journals for the betterment of clinical research.

I am also aware of my right to opt out of the trial at any time during the course of the trial without having to give the reasons for doing so.

Signature /thumb impression of the patient :

Date :

Name of the patient :

Signature of the investigator :

Date :

Head of the Department :

தேசிய சித்த மருத்துவ நிறுவனம், சென்னை-47.நோய் நாடல் துறை

“அதி நுரை நீர்நோய் கணிப்பு முறைமற்று ம் குறிகுணங்களை பற்றிய ஓர் ஆய்வு”

ஒப்புதல் படிவம்
ஆய்வாளரால் சான்றளிக்கப்பட்டது

நான் - ந்த ஆய்வை குறித்த அனைத்து விபரங்களையும் நோயாளிக்கு புரியும் வகையில் எடுத்துரைத்தேன் என உறுதியளிக்கிறேன்.

தேதி :
கையொப்பம் :
- டம் :
பெயர் :

நோயாளியின் ஒப்புதல்
நான், என்னுடைய சுதந்திரமாக தேர்வு செய்யும் உரிமையைக் கொண்டு - ங்கு தலைப்பிடப்பட்ட “அதி நுரை நீர்” நோயை கணிப்பதற்கான மருத்துவ ஆய்விற்கு என்னை உட்படுத்த ஒப்புதல் அளிக்கிறேன்.

என்னிடம் - ந்தமருத்துவ ஆய்வின் காரணத்தையும், மருத்துவ ஆய்வுக்கூட பரிசோதனைகள் பற்றி திருப்தி அளிக்கும் வகையில் ஆய்வு மருத்துவரால் விளக்கிக் கூறப்பட்டது.

நான் - ந்த மருத்துவ ஆய்வின் போது காரணம் எதுவும் கூறாமல், எப்பொழுது வேண்டுமானாலும் - ந்த ஆய்விலிருந்து என்னை விடுவித்து கொள்ளும் உரிமையை தெரிந்திருக்கின்றேன்.

தேதி :
- டம் :
கையொப்பம் :
பெயர் :
தேதி :
சாட்சிக்காரர் கையொப்பம் :
பெயர் :

உறவுமுறை :

DEPARTMENT OF NOI NAADAL

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47

FORM IV- A -PATIENT INFORMATION SHEET

PURPOSE OF RESEARCH AND BENEFITS:

The diagnostic research study in which your participation is proposed to assess the diagnostic methods in Siddha methodology and neikuri examination in Athi Nurai Neer patients. Knowledge gained from this study would be of benefit to patients suffering from suchconditions for the diagnosis and prognosis.

STUDY PROCEDURE:

You will be interviewed and examined as OP and IP patients at the study centre. At the first visit the physician will conduct a brief physical examination and assess the condition followed by Envagai thervu and routine blood and urine analysis. After matching the inclusion criteria you will be included in this study and you will be examined on the basis of Envagai thervu.

POSSIBLE RISK:

During this study there may be a minimum pain to you while drawing blood sample.

CONFIDENTIALLITY:

Your medical records will be treated with confidentiality and will be revealed only to other doctors / scientists. The results of this study may be published in a scientific journal, but you will not be identified by your name.

YOUR PARTICIPATION AND YOUR RIGHTS:

Your participation in this study is voluntary and you may be withdrawn from this study anytime without having to give reasons for the same. You will be informed about the findings that occur during the study. If you do agree to take part in this study, your health record will need to made available to the investigators. If you don't wish to participate at any stage, the level of care you receive will in no way to be affected. The Ethics committee cleared the study for undertaking at OPD and IPD, NIS. Should any question arise with regards to this study you contact following person

P.G student:
Dr. Gayatri.R. 1st Year
Department of Noi Naadal
National Institute of Siddha
Chennai-600 047.

தேசிய சித்த மருத்துவ நிறுவனம், சென்னை-47.
நோய் நாடல் துறை

"அதி நுரை நீர்நோய் கணிப்பு முறை மற்றும் குறிகுணங்களை பற்றிய ஓர் ஆய்வு"

நோயாளியின் தகவல் படிவம்

ஆய்வின் நோக்கமும் பயனும்:

தாங்கள் பங்கேற்க்கும் இவ்வாய்வு"அதி நுரை நீர் "நோய் கணிப்பு முறை மற்றும் குறிகுணங்களை பற்றிய ஓர் ஆய்வு" சித்த மருத்துவ முறையில் நோயை கணிப்பதற்கான ஓர் ஆய்வுமுறை.- வ்வாய்வு தங்களின் நோய்கணிப்பை பற்றியும் நோயின் போக்கைபற்றியும் அறிய உதவும்.

ஆய்வு முறை:

தாங்கள் நேர்காணல் மற்றும் பரிசோதனைகளின் மூலம் உள்நோயாளி, வெளிநோயாளி பிரிவில் ஆய்வு செய்யப்படுவீர்கள். முதல் நேர்காணலின்போது ஆய்வாளரால் உடல் பரிசோதனை, நீர், - ரத்தம்,மற்றும்மலம் பரிசோதனை செய்து குறிப்பிட்ட குறிகுணங்கள் - ருப்பின் - வ்வாய்விற்காக எடுத்துக்கொள்ளப்படுவீர்கள்.

நேரும் உபாதைகள்:

- வ்வாய்வில் - ரத்த பரிசோதனைக்காக - ரத்தம் எடுக்கும்போது சிறிதளவுவலி ஏற்படலாம்.

மந்தணம் :

தங்களின் மருத்துவ ஆவணங்கள் அனைத்தும் மருத்துவர், ஆய்வாளர் அல்லாத பிறரிடம் தெரிவிக்கப்படமாட்டாது.

நோயாளியின் பங்களிப்பும் உரிமைகளும்:

- வ்வாய்வில் தங்களின் பங்கேற்புதன்னிச்சையானது. - வ்வாய்வில் தாங்கள் ஒத்துழைக்க - யலவில்லையெனில் எப்பொழுது வேண்டுமானாலும் காரணம் எதுவும் கூறாமல் விலகிக்கொள்ளலாம். - வ்வாய்வின்போது அறியப்படும் தகவல்கள் தங்களுக்கு தெரிவிக்கப்படும். நோயாளியின் ஒப்புதலுக்கிணங்க நோய்கணிப்பு விவரங்களை ஆய்வாளர் பயன்படுத்திக்கொள்வார். நோயாளி ஆய்வினிடையே ஒத்துழைக்க மறுத்தாலும் எந்த நிலையிலும் நோயாளியை கவனிக்கும் விதம்

பாதிக்கப்பட மாட்டது. நிறுவன நெறிமுறை குழுமம் (Institutional Ethical committee) மேற்கண்ட ஆய்வினை மேற்கொள்ள ஒப்புதல் அளித்துள்ளது.

ஆய்வு குறித்த சந்தேகங்கள் - ருப்பின் கீழ்கண்ட நபரை தொடர்பு கொள்ளவும்.

பட்டமேற்படிப்பாளர் :

மரு.காயத்ரி.ர(முதல்வருடம்)
நோய் நாடல் துறை
தேசிய சித்த மருத்துவ நிறுவனம்,
சென்னை-47.